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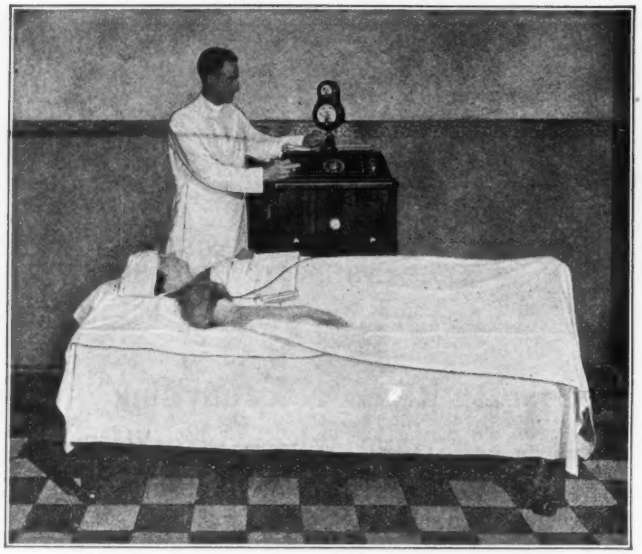

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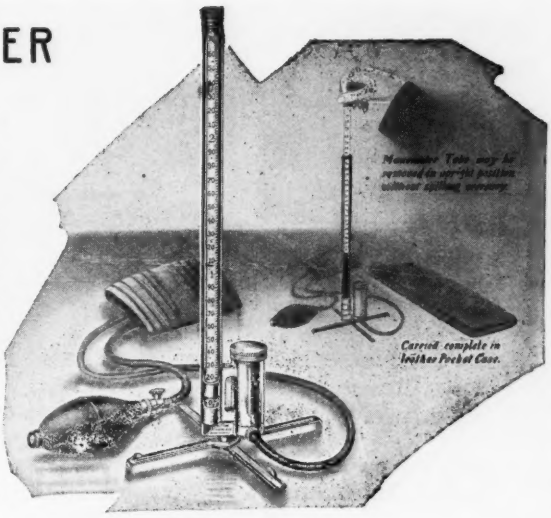

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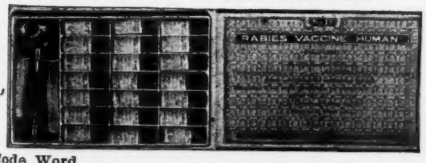

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THE JOURNAL
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VOL. XXVII TOPEKA, KANSAS, MAY, 1927 No. 5

Free Full-Thickness Skin Transplantation

EARL C. PADGETT, M.D.
Department of Surgery

Read before the Wyandotte County Medical Society, February 15, 1927.

HISTORICAL

Centuries ago in old records concerning the tile-maker's caste in India a description is recorded of the free transplantation of full thickness skin from the gluteal region to the nose. Previous to transplantation the gluteal region was beaten, producing hyperemia. For the purpose of advertising an ointment early in the nineteenth century, two charlatans, one of whom was a woman, Gamba Curta, cut a piece of skin from their bodies, passed it around to spectators for inspection, then replaced the pieces on their original raw bed and applied their ointment with a dressing. It is related that the grafts "took" and healed with little scarring. Baronio (1804), a physiologist, aroused by the knowledge of this exhibition, experimented on a sheep's tail and successfully transplanted full thickness skin from one side to the other.

As early as 1823, Bunger of Marbury successfully transplanted a free piece of whole skin from a woman's thigh to her nose and again in 1843, J. Warren Mason, one of the famous surgeons of Boston, successfully covered the alae of the nose with a bit of detached full thickness skin from the arm. Netolitchi of Germany (1869), Lawson of London (1871), Le Fort of France (1872) undoubtedly preceded Wolfe of Edinburgh in 1875, in the successful transsplantation of whole skin. Wolfe, whose name has been connected, along with Krause of Germany, with this particular type of skin grafting, used the method for an ectropion of the eye-lid. The following year, Krause (1876) reported 21 cases at the 22nd German Surgical Congress. Now the grafts are often designated Wolfe-Krause grafts.

Thus, although isolated records of an occasional success are found here and there in the literature many years previous to Wolfe and Krause, the method was never emphasized, previous to the articles of Krause the significance of the method was not appreciated. Even these men and of course those preceding them used only small bits of skin and it remained for the method of successfully transplanting large areas — as large as 6x10 inches to be developed as recently as after the late World War—after which every known possibility in repair work was revived and if possible developed.

During the three years following the war Blair of St. Louis was especially interested in plastic repair work and during that time, a technique was developed which allowed one in an aseptic field to confidently expect at least a 90% "partial take" —irrespective of the size of the graft or its location. It had been known that these grafts usually grew best on the forehead. Here, it is apparent, one has a good smooth firm background on which to obtain uniform pressure on aplication of the ordinary dressing. Moreover, the observation has been made that a certain tension supposedly to open the minute endothelial spaces of the skin was an aid to a good "take."

Finally it became apparent that the following five factors seemed very significant to success.

(a) That the skin should be cut in a manner that left no fat attached.

(b) That a sterile field markedly increased one's chances of success.

(c) That a dry field without oozing of blood or blood clot in which all vessels were tied without massing of tissue with the finest possible suture material was important.

(d) That slight tension in all directions was of some consequence.

(e) That it was absolutely essential that moderate pressure and fixation be maintained to cause contact with the raw base —even in all the various grooves and depressions.

Previously, all five of these prerequisites to success had been emphasized but the known technique of obtaining moder-

ate pressure and fixation was often faulty. The last condition was finally more satisfactorily obtained when Blair thought of the old marine sponge which had been dropped largely from the surgical armamentarium. A large marine sponge of fine texture when damp and under compression gives a soft resilient surface pressure to an underlying uneven base and later when dry maintains a fixation somewhat similar to a cast.

As confidence of success became more certain the raw surfaces to be grafted became larger and larger until any surface was attempted, (which did not endanger the patient's life or for which skin could be obtained) with confidence in obtaining a successful result.

TYPE OF CASES

One's imagination is immediately stimulated when he pictures those cases in which a successful technique of full thickness skin transplantation is applicable.

Possibly the widest and most brilliant application includes those cases of marked cicatricial contracture following burns so familiar to surgeons on the staffs of our children's hospitals. Although the proper application of thin Thiersch grafts or "pinch grafts," along with the proper maintenance of position do much to promote a happy result, it is often impossible in the more severe burns to prevent marked contractural deformity. From the standpoint of appearance these scars are a real calamity, and when involving the neck, the axilla, the elbow, the knee or the hand, may almost totally destroy function.

In hand contractures due to scar, one can get some very remarkable results even when the tendons are just at the surface and have to be laid bare at operation. In many cases, the result is esthetically more pleasing than the old flap transplant and from the standpoint of function offers in cases without tendon loss a perfectly satisfactory result.

Ectropion due to scar of the external eye-lids or an eversion of the lids are usually best handled with the Thiersch inlay graft because it is difficult here to get a clean field for a Wolfe graft.

Certain selected cases of roentgen-ray burns with ulceration, pain, and possibly slight contracture can be relieved by excision followed by full thickness skin grafting if the destruction of soft tissue is not too deep. In the latter cases a flap transplant or a combination of a flap and a full thickness skin graft, are usually the graft methods of choice.

Hemangiomas of the face not involving the eye lids in adults offer possibilities by treatment with excision followed by whole thickness skin transplantation.

Such congenital anomalies as complete fusion of the fingers offer a very brilliant field for the application of this principle. In one operation the whole situation can be corrected as far as the soft tissues are concerned. In these cases full thickness skin transplantation offers a great advantage over pedicled flaps.

Very satisfactory eye brows can be made from the hair bearing scalp skin.

TECHNIC

The scar contractures are laid wide open as soon as a clean field is obtainable without regard to the size of the resulting raw surface, providing that the denuded surface is not of such extent as to jeopardize the life of the patient and that enough skin to cover the area can be obtained. The skin is cut by pattern from the abdomen, back or thigh, in such a manner that no subcutaneous fat remains, after which the skin is transferred to the denuded area. Hemostasis should be almost perfect. The transplant is sewed in place on a slight tension. A dressing is applied consisting of a thin layer of gauze impregnated with a mild antiseptic ointment on which is super-imposed one or many large marine sponges. The sponges are bandaged just firmly enough to get good approximation between the graft and the underlying tissue and not tight enough to cause pressure necrosis. This original dressing is not removed until eight or ten days have passed. On removal of the dressing the stitches are taken out and a moist saline gauze dressing is applied three to five times daily. The gauze is always kept moist.

When one removes the first dressing, in about 50% of the cases there will be seen a few superficial blisters. These are treated best in about the same manner that one treats a simple blister due to a burn that is, let it rupture when it gets ready, providing the contained fluid is clear. If the fluid in the bleb becomes purulent it should be opened immediately.

ULTIMATE RESULTS

In planning the operation one takes into consideration the fact that the graft will contract about one fourth and opens up the tissues very completely, over correcting

FIG. I.

FIG. II.

Figures 1 and 2 presents a case of cicatrical contracture due to an extensive burn, showing the front and side views before operative procedures were undertaken.

FIG. III.

FIG. IV.

Figures 3 and 4 (Same case as Fig. 1 and 2) now has a pedical flap beneath the chin on both sides. The large area from above the hyroid region down over the sternum shows a full thickness graft about three weeks after application. His head is now well loosened up. This boy will return after six months for final excision of unsightly scars.

the deformity as much as possible. In situations like the neck, the axilla and the fingers, the maximum amount of extentsion or flexion is obtaned, as the case may require, by cutting across or excising all scar tissue bands that are put on tension by over correction.

Blair in 1924 presented 106 cases before the American Surgical Association, practically all of which I saw. These cases included those in the developmental stage of the technique and showed an average per centage of success of 77.41 per cent. 58 per cent of these cases showed a 90 per cent "take". The average size of the transplants was 8.43 square inches. Some of these were fully as large as two hands.

The new transplant may be somewhat shiney and smooth. I once made some twenty microscopical sections of a series of these grafts removed at different periods after transplantation. The corrugations of the superficial epithelial surface which one sees in normal skin sections are absent. The epithelial surface in these sections was perfectly smooth. It was also noted that the elastic tissue of the skin showed degenerative changes. I concluded that the shiney and smooth appearance of some of these transplantations was caused by an absence of the normal skin corrugations. Degeneration of the elastic fibers of the transplant with some increase of fibroplastic tissue could explain this phenomena. Some variation is seen in the surface texture. A large majority that have shown a good "take" without superficial blistering or much superficial exfoliation of the epithelium show grossly practically normal skin texture.

Certain grafts show a tendency to pigmentary changes, usually more marked in the brunette. These pigmentary changes may be uneven and blotchy and may lessen materially the esthetic result if the graft has been applied to the face. As a rule, the younger the patient the better the "take."

Even when the blisters become infected and the superficial squamous epithelial layer is cast off, if the infection is stopped the surface of the deeper layers may become covered with epithelium and give functionally a very satisfactory result; altho this occurence increases the tendency to future shrinkage. These damaged areas usually remain more pigmented and darker in color, varying in appearance from the non-damaged part of the graft, somewhat as epitheliated scar differs in appearance from normal skin.

The majority of the hairs and hair follicles in areas that have "taken well" grow nearly normally so that in transplantation, one should not use hair bearing skin unless hair is desired.

After four to six weeks a thin layer of adipose tissue forms beneath a full thickness graft and the skin loosens from the underlying tissue, becoming movable. Thiersch grafts do not do this to any great extent. Beginning about six weeks after transplantation a new nerve supply starts in from the edges. First, tactile sensibility, then pain and last temperature sense appears. A large graft may not regain sensation for more than a year. A depressed area successfully grafted fills up to the level of the normal surroundng skin after some months.

DISCUSSION

The main advantages of the full thickness graft are (1) that contraction is markedly less than following a Thiersch graft (A Thiersch graft does not give a satisfactory result in these markedly deformed cases); (2) that in the palm of the hand, for example, and other areas subjected to considerable trauma, full thickness grafts will give a good functional result if there is any soft tissue between the graft and the bone; (3) that in certain congenital anomalies as, for instance, web fingers it offers a method giving a good result in one operation; (4) that in certain disfiguring facial blemishes it has possibilities not possessed by other types of skin transplants.

If the bone is nearly bare or thickness is desired a flap transplant offers advantages not otherwise obtainable. The disadvantage of a flap is first, that its thickness in certain areas gives a clumsy result less pleasing from the esthetic standpoint, and second, that several operations are required. The Wolfe graft can be transplanted usually in one or two operations. The flap transplant however, has its distinct indications and probably is more useful in the various phases of plastic surgery than the full thickness graft. The Thiersch graft has the advantage that it will grow in an unclean field. From the standpoint of appearance a full thickness graft in successful cases is far superior to the Thiersch graft which is generally pale and white. The Wolfe graft in good "takes" appears, eventually, as normal skin.

NOTE — I am indebted to Dr. T. G. Orr for the case illustrated. The pedical flaps had been very successfully transplanted by Dr. Orr before I saw the case.

The Management of Bow Legs, Knock Knees and Flat Feet in Children

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Often I have been impressed with the fact that many doctors are at a loss to know how to advise parents who present their young children for the consderation of faulty conditions of their legs and feet. At the outset of the discussion I wish to say that not always is it possible to determine at once the proper procedure; but there is a definite plan to follow in the management of these cases. No one can tell whether or not a child with bow legs will itself correct this condition; yet it is an indisputable fact that many children do. However, very few children with knock knees will improve without aid; every child with a positive knee valgus should be given attention; and flat feet in children should be thought of as weak feet.

Bow legs or knock knees never are caused by the normal child walking too soon, but are the result of the bones being too soft; thus suggesting faulty nutrition. If the child is under-nourished attention to this condition is just as important as attention to the deformity.

In the management of cases of bow legs, one should keep in mind that in children under three years of age whose nutrition is subnormal, usually it is possible to secure correction by proper diet and hygiene combined with daily manipulative measures executed by the parents.. Certainly these means should be tried and the case observed. In the cases that are well nourished or in those where the parents have bow legs, conservative measures may be tried, but the likelihood of success is not great. In either of the above mentioned types in cases of children under three years of age, when it has been determined that the bowing has not decreased, braces should be applied. The braces are all similar; the principle being to pull the leg into the corrected position. (See illustration No. 1).

After a child has passed the age of four years and has a considerable degree of bow leg deformity, braces will not effect a correction. The proper treatment at this time is to perform an osteotomy and correct the bowing as well as the rotation that usually is present; then, put the legs in a double plaster of paris cast for six weeks. At the end of this time, the cast should be removed and measurement made for braces. The cast should be re-applied until the braces are ready for wear, at which time the child should be permitted to run about in braces for a period of six months to a year. If braces cannot be obtained, then the casts should be re-applied and changed at intervals but kept on for four or five months and the child encouraged to be active. It is necessary for these cases to have the protection of braces or plaster for this length of time on account of the knee joints. The correction of the bone does not rectify the weight bearing lines of the joints. This comes with weight bearing and better results will be secured with protection.

Illustration No. 1—Simple bow Leg braces applied.

Needless to say ,in cases of bow legs careful attention should be given to the diet. These cases need fats, such as cream, butter, bacon and soups. They should also have cod liver oil routinely. Early attention to

these dietetic factors will correct the condition in a large per cent of cases.

Knock knees are similar in origin to bow legs; but unlike bow legs, do not tend to correct themselves spontaneously. In children under three years, the inner sides of the heels and soles of the shoes should be raised one quarter of an inch and daily manipulation and general attention to the diet as suggested for bow legs should be instituted. In a large per cent of cases, these measures will prove eminently satisfactory; and it is absurd to submit such patients to operation. However, as was suggested in discussing bow legs, there are a few cases that will require braces and some that will require an operation. Those that seem to inherit the condition, or those that are of the heavy type usually do not respond to palliative measures. In the heavy

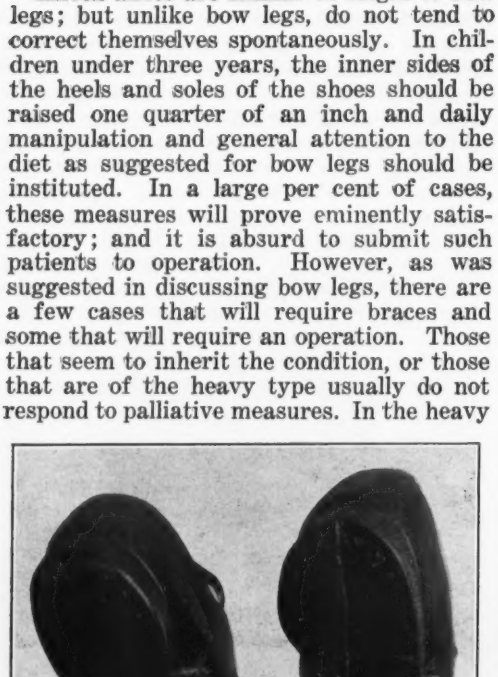

Illustration No. 2—Inner edges of heels and soles raised one-quarter inch. This alteration should be made for children with weak feet or with knock knees.

types endocrine disturbances should be thought of. As in bow legs after the child has passed four years of age, operative measures (usually a supracondylar osteotomy), followed by plaster casts and braces for six months or more are required.

The manipulation method that I employ for knock knee is as follows: With the child lying on a bed or table, the knee is grasped and held firmly in one hand; with the other hand, the ankle is grasped and a slow gradual pressure is made on the knee, relaxing it at intervals and then increasing the tension until the deformity is over corrected, holding it in this position for a time, and repeating the performance for the other leg. For bow legs, the procedure is practically reversed, except that the heel of the hand that grasps the knee is allowed to come into contact with the outer apex of the bow. In cases of either knock knees or bow legs, the mother or father of the patient is instructed to perform this manipulation at least twice a day.

In regarding flat feet, this discussion will be limited to the so-called idiopathic type and no consideration will be given to the acquired forms resulting from paralysis or other diseases or injuries.

The feet of all infants are flat primarily. At least they have the appearance of being flat, due to the fat pad that normally is present along the plantar fascia. When the child begins to walk, the entire sole of the foot is in contact with the floor. This permits better balancing and so assists the child in learning to walk. If the muscles are balanced properly and maintain a normal tone, the feet gradually assume an arch, due to the absorption of the fat pad. If, however, from any cause the muscles relax, the foot will pronate or roll into valgus as the fat pad is absorbed and so becomes a weak foot. In this condition, the natural instinct of the lay mind is to put high shoes on the child. This further weakens the foot muscles and thereby tends to insure a corresponding increase in the deformity.

The case of flat or weak feet should be treated by putting the patient into low shoes, thereby allowing free action of the ligaments and foot muscles, thus stimulating development. The heels and soles of the shoes should be raised by wedge-shaped pieces of leather being placed along the inner border of each, the same as for knock knees. (See illustration No. 2). Usually, one quarter of an inch in thickness is sufficient to roll the foot out into the proper position of weight bearing. Occasionally, a felt pad placed under the posterior arch of the foot is required to secure the proper balance. If these children be permitted to go bare footed a portion of the time, a great improvement in the muscle tone will be noted. This also is contrary to the general belief and some parents are reluctant to permit it.

Arch supports and rigid shoes not only limit the muscle action but restrict the blood supply, thereby causing further loss of muscle tone and atrophy, preventing the maintenance of the proper balance of the foot, even after re-molding of the bones has

been accomplished. Therefore attention to nutrition and activity is just as essential in weak feet as in bow legs or knock knees. Occasionally operative procedures are required for the rectification of bony deformities in congenital flat foot, but such cases are extremely rare; and, after the bony deformity has been righted, correct posture can be maintained only by proper muscle balance and tone.

In conclusion I wish to say that it is the duty of the doctor when these cases present themselves to interest himself first, in the general nutrition of the child; second, in seeing that proper shoes with alterations are secured; third, that the child be kept under observation and managed as indicated.

The Malaria Treatment for Neurosyphilis

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The limited space here forbids much more than a resume of this subject. A somewhat complete treatise on therapeutic malarial inoculation necessarily would cover a great many pages. The conclusions of the author are drawn from an analysis of a fairly voluminous literature and his own cases treated during the past two years. If the reader wishes to study more minutely the various phases of this treatment, he may be referred to Wagner von Jauregg, Gerstmann, and Ferraro and Fong and many other articles. Gerstmann in his monograph of 229 pages, and Ferraro and Fong have appended excellent, large bibliographies.

From time to time, covering a period of more than fifty years, foreign proteins and bacterial toxins have been used in the treatment of organic diseases of the brain as well as some purely functional psychoses. Several personal experiences with incidental complicating infections, producing apparently beneficial results in the course of some neuropsychiatric disorders, might be cited. Tuberculin, typhoid toxin, the organism of relapsing fever and the malarial parasite in particular might be mentioned.

Jauregg began his experimental work with foreign proteins in 1887, and by his work first reported in 1917, is responsible for the use of the tertian malarial organism in the treatment of paresis as seen today.

Only the tertian type of plasmodium has been used. This holds largely because the parasite is so readily controlled, being promptly destroyed by appropriate doses of quinine. It is something of a problem to find a proper source for inoculation. In the state of Kansas malaria is found only in a few counties and then to a limited extent.

Three principal sources have been employed for the inoculation. 1. Anopheles mosquitoes carrying the tertian malaria are seldom used for obvious reasons. 2. Non-syphilitic patients with tertian malaria would make the ideal source for obtaining the organism. But malaria is seldom found in many states, and even in the southern states the disease is not so common compared with a generation ago, thanks to medical science and modern sanitation. Comparatively few patients in this country are inoculated by this means. 3. The vast majority have the plasmodium introduced by using the blood of a case of paresis or other metaluetic disease under malaria treatment. Accuracy in the diagnosis is indicated. We do not believe that a case of active metaluetic disease is susceptible to a new syphilitic infection. Furthermore it is not expected that spirochetes thrive or live in blood loaded with tertian malarial organisms and their toxins. No quinine or arsenicals should be used before or during the course.

METHOD OF INOCULATION

A quantity of blood ranging from 1 to 7 c.c. is removed from an arm vein, using an ordinary intravenous needle and syringe. Take it from the donor preferably at the height of or shortly following a malarial paroxysm. This gives the highest percentage of successful inoculations, although blood taken at any hour might furnish the desired results. If possible the donor and recipient should be close at hand so that the transfer may be carried out quickly. If the blood is to be held for any length of time it should be citrated and kept at a temperature of about 98 degrees Fahrenheit. The longer the blood remains outside of the body the smaller will be the percentage of successes. Proper aseptic precautions should be exercised at all times. The blood may be introduced by the subcutaneous, intramuscular or intravenous routes. If by the intravenous method, use proper precautions relative to coagulation. The intravenous method probably is more certain, and reduces the incubation period about three days.

SYMPTOMS AND COURSE

The symptoms and course for tertian malaria varies considerably. The incubation period ranges from 2 to 30 days. Much emphasis is placed on the temperature.

Even this is frequently uncertain. From the same strain we may have the typical paroxysms every other day or every day. Quite irregular temperatures frequently are encountered. It may range from 102 to 106 degrees F. for three to five hours every other day. Typical chills occur in about fifty per cent of the cases. Wtih the paroxysm an increased pulse and respiratory rate may be observed. Other toxic manifestations appear in a few days. These include general malaise, increasing weakness, impaired appetite, reduction in weight, coated or dry tongue, and especially herpes labialis. One of my cases had the herpetic eruption on the lips with very little increased temperature at any time.

All clinicians appreciate the value of the examination of the blood of a suspected patient for malaria, and particularly to determine the type. However, it may not always be easy to find plasmodia in every case and at every stage even though we suspect their presence. It is possible that they could be found in the internal or glandular organs and not in the peripheral circulation where the blood has been removed for staining. During the course there is a gradual destruction of the red blood corpuscles with a definite and at times marked reduction in the hemoglobin. This feature has some prognostic value, and is a guide as to when to check the treatment.

WHEN TO STOP THE TREATMENT

We do not always agree with the Irishman who believing that he got some benefit from the small dosage directed by the physician, and thinking that an increased dosage would produce a parallel improvement, proceeded to use a strikingly toxic dosage with serious results. However, it is believed that a maximum amount of the malaria treatment should be allowed the patient. Here is opportunity for good scientific judgment by the therapeutist. Each patient should be treated as an individual. Some writers advocate a certain number of chills, frequently giving figures ranging from eight to fifteen. We cannot be guided solely by the number of malarial paroxysms. In the first place some patients will tolerate much more than others for several reasons. I am guided partially by the number of chills, the tolerance of the individual patient. the various toxic effects, and especially the reduction of the number of erythrocytes and hemoglobin percentage. I have had two patients where it was necessary to stop with six paroxysms. Again one of my cases tolerated quite well twenty-two definite ones. As indicated above, it is not always possible to count exactly the number of chills.

HOW TO CHECK THE MALARIA

Quinine is a real specific for tertain malaria. It is merely a matter of selecting the dosage. With all of my cases it has been found quite satisfactory to give the medicine by mouth. The dose for an adult usually ranges from one half to one gram three times per day. A course of quinine treatment for at least one week is essential in all cases. In some it is necessary to give the quinine over a period of ten to fifteen days. A repetition of the treatment is required infrequently. If there is any question about the cessation of the malaria, a few doses of neoarsphenamine may be given. Arsenic too will destroy plasmodia, to say nothing about the spirocheticidal possibilities. An illustrative case may be cited briefly. A patient with paresis treated at Bell Memorial Hospital had tolerated fairly well some twelve indefinite paroxysms. He was markedly toxic with severe labial herpes, and had a fair reduction of the erythrocyte count and hemoglobin. Suddenly there appeared a marked myocardial weakness. At once he was given one half gram doses of quinine sulphate by mouth three times per day. Severe chills ceased at once. However, he frequently had about a half a degree of temperature during the first week of treatment. Then the dosage was increased to one gram which was tolerated well and which checked the fever promptly.

DANGERS

The immediate dangers may be divided into two groups: (1) complications due directly to the malaria and (2) mortality. The first is of lesser importance. A few minor complications can be taken care of readily.

The direct mortality rate resulting from the treatment warrants careful consideration. No patient, relative or guardian should be told that the treatment is harmless. In a review of the literature, and selecting at random 1019 cases, I find an average mortality rate of 13.7 per cent. Mortality rates ranging from 2.5 per cent to 28.4 per cent are noted. 32 personal cases observed have had a mortality rate of 9.4 per cent. Had I desired to maintain a low mortality rate, two of my fatal cases never would have been selected for the treatment. One was that of a physician with tabo-paresis, and complicated with nephritis and

other troubles. He himself and his medical advisers desired the treatment despite the great danger. Another was a man with well advanced tabes, with some early paretic manifestations, and in extremis physically. I feel that the deaths can be kept below 5 per cent by a judicious selection of cases. Reflect on past prognosis for paresis. Nearly all these patients have died within from one to five years following a certain diagnosis. Most of the paretics have been a great burden and a dangerous menace to society and themselves. Therefore, the mortality rate quoted is comparatively insignificant.

SELECTION OF CASES

The malaria treatment may be used for paresis, tabo-paresis, tabes, metaluetic optic atrophies, chronic degenerative syphilitic myelitis, and a few other uncommon types of late syphilis. In selecting cases of paresis and tabes, avoid those with much nephritis, serious myocardial or aortic troubles and objectionable states of inanition. Never can we hope to restore brain functions where many neurons have been destroyed with a concomitant extensive gliosis, by any treatment. The very early stage of paresis is the choice one.

RESULTS

It is certainly too early to draw final conclusions relative to what may be accomplishsed by this new method of treatment. An analysis including some 3500 cases reported in the literature and my own, showed a wide variation in the results. For paresis satisfactory remissions vary from 8 to 50 per cent. Perferably should we wait at least one year from the time of the treatment before final conclusions are made. Ultimately a five or ten year period will be interesting. We have no good practical mechanical scale by which to judge all the cases. Individual interpretations vary considerably. Gerstmann with 400 cases, reports 33.2 with good results. Kirby gives 35 per cent. It is not always an easy matter to follow up cases year after year. I have four patients suffering from paresis who were given the malaria treatment and whom I have been able to observe adequately for a long period. These men are doing a certain amount of work and getting along in society fairly well. Without the malaria treatment I feel that each man would be dead or locked up in some institution today. Tabes compared with paresis seems to offer less favorable possibilities for therapeutic malaria.

GENERAL SUMMARY

1.—Today the malaria treatment for metaluetic disorders, and especially paresis, vies with tryparsamide. Comparative values five or ten years hence will be interesting.

2.—Certainly many cases of paresis have shown remarkable improvement with malaria; and that did not yield to a number of other therapeutic methods or courses, including iodides, mercury, bismuth and the various arsenicals.

3.—Other therapeutic measures and hygienic attention is indicated for a long period following the course of malaria.

4.—Second or third inoculations with the tertian parasite may be indicated after a proper interval.

5.—Especially would I admonish that careful physical, neurological, psychiatric, and adequate spinal fluid examinations be made before considering this treatment.

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Hypertonic Salt Solution in the Treatment of Superficial Infections

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Since the Lister period of antisepsis, we have all fallen into the habit of depending upon chemical germicides in our treatment of superficial infections, granulating surfaces, sinuses, abscess cavities and similar lesions. We have used and discarded one after another of them from carbolic acid to mercurochrome which at present is preeminent in the field. They have all fallen short of the performance we have expected of them. The reason they have done so is that they can kill only those bacteria with which they come into contact. The bacteria lying deep in the inflamed walls of these lesions are the ones causing the disturbance and as yet no germicide has been discovered which has a penetrative power sufficient to reach them unless it is used in such strength as to cause it to kill living tissue cells as well as bacteria.

The theory upon which the use of hyper-

tonic salt solution is based compels our attention. We know that non-colloid bodies in solution pass through semi-permeable membranes in the direction of the fluid exerting the higher osmotic pressure. So when we remember that the cells forming the walls of abscesses, granulating surfaces and sinuses constitute a semi-permeable membrane, it follows that when we place in contact with this membrane, a hypertonic salt solution, there must be an excursion of the cell plasma and of the lymph surrounding the cells in the direction of the hypertonic solution. This reversal of the direction of the lymph flow must inevitably carry out with it toxins and living bacteria that have been lodged in the depths of the inflammatory wall surrounding the lesion. In other words, the toxins and bacteria are, by these physical means, removed from the tissues where they are doing damage and carried into the dressing where they are harmless. It doesn't matter whether the bacteria are killed or not.

This plan of treating infected lesions was first given wide prestige by the medical department of the British army, Sir Almoth E. Wright and A. E. Morrison having worked out its details. The results gotten in the management of the badly infected war wounds were excellent.

Various substances such as sodium chloride, sodium citrate, glycerine, glycerite of boroglyceride, glucose, and magnesium sulphate in a variety of combinations have been used, but the principle of their action is the same. It would seem that magnesium sulphate has certain advantages over other salts as it is less painful, exerting as it does a local anesthetic effect; it is not readily absorbed; it definitely inhibits bacterial growth and it is easily obtained at a low cost.

I have used this plan in treating flat granulating surfaces following burns, perionychia, lacerated wounds, infected compound fractures, osteomyelitis after osteotomy has been done, fistulous tracts, chronic empyema cavities, chronic endocervicitis, cellulitis, boils and carbuncles. The results have been uniformly satisfactory.

The technic of treatment differs slightly with the character of the lesion. For boils, carbuncles, perionychia and flat granulating surfaces, it is best to use a paste made in the following manner: Heat a mortar and pestle. Place a quantity of glycerine in the mortar and gradually stir into it dried magnesium sulphate until a thick paste is formed. This must be kept in an hermetically sealed container until used as the mixture rapidly absorbs moisture from the air. It is applied to the lesion and the dressing is covered with a piece of oiled silk.

Another technic I have used in the treatment of boils and carbuncles in which the necrotic area is already pointing, is to destroy the pellicle of skin with 95 per cent phenol on a small wooden applicator thinly covered with cotton letting it form a small tunnel into the necrotic area. Usually a little pus escapes but it is important to refrain from squeezing the inflamed tissue as squeezing tends to break down the protective barrier of leucocytes which has been thrown up about the nidus of infection. Gently fill this tunnel with crystals of magnesium sulphate. In twelve or twenty-four hours, you will find that considerable pus and lymph have drained into the dressing and the tunnel is much larger and will take a larger quantity of crystals. This process is repeated until the slough has separated and the lesion presents a clean healthy granulating wall. It is especially amazing to note how rapidly the general symptoms of toxemia subside after this treatment is begun; how rapidly the slough separates and the wound fills in with granulations, and how relatively slight the resultant scar is.

For sinuses too small to permit the introduction of the paste, and in large abscess cavities like chronic emypema of the pleura, I prefer to use a solution made after the lowing formula:

Magnesium sulphate	50 parts
Glycerine	10 parts
Boiling water	40 parts

On cooling, the salt recrystallizes from this supersaturated solution; so it is necessary to heat it in a water bath each time before using.

———R———

Cholecystectomy in Gall Tract Disease

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Any logical treatment of the gall bladder should be based upon a knowledge of the embryology, anatomy, physiology and pathology of the entire gall tract. Since the liver, gall bladder, gall ducts, duodenum and pancreas all arise from the same embryonic intestinal tract bud it is reasonable to expect that these organs may be associated in their functions and their diseases. Such is certainly true and diseases of the gall bladder should always be considered a part

of a possible disease of any or all of the above organs.

In the presence of established disease of the gall tract what is the proper surgical treatment? This must obviously be answered from the standpoint of the various types of diseases of this tract and must take into consideration the general condition of the patient. It is my present opinion that, if consistent with operative safety, cholecystectomy should be done in all infections of the gall bladder serious enough to require surgery of this organ. I hope to support this opinion in the brief discussion which follows.

When an abdomen is opened and the gall tract examined what are the cardinal points which indicate to the operator that surgery of the gall bladder is advisable? It is quite well known to those who do considerable upper abdominal surgery that the gall bladder at times shows very little gross evidence of pathology in spite of definite history and physical findings of gall tract disease. There are five definite signs of pathology of the gall bladder which should be kept in mind whenever this organ is examined at the operating table. They are (1) evidence of chronic hepatitis, (2) thickening of the gall bladder wall with change in color from the normal slaty blue, (3) presence of gall stones, (4) adhesions to the gall bladder and (5) enlarged lymph node at or near the junction of the common and cystic ducts. If these signs are kept in mind a gall bladder will rarely be needlessly removed.

Some of the arguments that have been presented favoring cholecystostomy are as follows:

1. It is a safer operation.
2. The gall bladder may be needed later if the common duct becomes obstructed.
3. Drains the infected gall tract.
4. Avoids dilatation of common duct which follows cholecystectomy.

Cholecystectomy is indicated for the following reasons:

1. It removes the diseased gall bladder.
2. Avoids recurrence of disease or stones necessitating another operation.
3. It prevents disabling adhesions from multiple operations.

There are undoubtedly some cases in which drainage of the gall bladder is indicated. In acute infections or empyema it is often unsafe to subject a patient to more than the minimum of surgical procedure to relieve the symptoms. This also applies to some cases of common duct obstruction in which jaundice is a marked symptom and liver damage is suspected. The pathology of a chronic infection of the gall bladder is found within the wall and not usually in the contents of the bladder. Simple drainage is not to be expected to effect a cure in all cases. If the infection is relieved by drainage the gall bladder tissue still remains and, like the appendix is prone to develop a recurrence.

Drainage of an infected gall tract is a rational procedure in acute infections. If there is evidence of acute cholangeitis associated with gall bladder disease or, if a tender liver indicates an active liver involvement, either cholecystostomy or a cholecystectomy with cystic duct drainage should be done. A. S. Lobingier of Los Angeles recommends drainage of the cystic duct in all cases of cholecystectomy in the belief that such drainage reduces the infection in the liver and pancreas.

The following reports indicate the futility of gall bladder drainage in some cases.

Case I.

Mrs. L. D. Age 41. History of gall tract disease 11 years. Drainage of gall bladder in 1919 with removal of stones. Second drainage in 1920 with removal of stones. Fistula persisted after this operation for two years. Cholecystectomy in 1925. Gall bladder found full of stones. This obviously a case in which the gall bladder should have been removed at the first operation. Gall stones found in the gall bladder three times.

Case II.

Mrs. M. L., Age 39. Exploratory operation for gall stones eight and one half years ago. No stones found and gall bladder not opened or removed. Three years ago gall bladder drained with some relief for one year. Present symptoms for two years with increase in severity five years before admission to hospital. Cholecystectomy in Jan. 1927. Adhesions very extensive in right upper quadrant of abdomen involving abdominal wall, colon, liver, gall bladder, duodenum and pyloric end of stomach. Evidently a peritonitis followed the cholecystostomy. Cholecystectomy was probably indicated at the first exploration eight and one half years ago.

There is a difference of opinion among surgeons in regard to abdominal drainage following cholecystectomy. Some believe that primary closure of the abdomen is perfectly safe if the operative technic has been good. It is undoubtedly true that a large percentage of patients will do well without

drainage, but the exception, with infection or leakage of bile, might prove disastrous. I believe that it is best judgment, especially for the occasional operator, to drain the abdomen in practically all cases after removal of the gall bladder.

The great danger of the discharge of infected bile within the peritoneal cavity is well known. McWilliams (Annals of Surgery, 1912) reported a death rate of 48 per cent in perforations of the infected biliary tract. In the experimental animal (dog) a biliary fistula draining directly into the peritoneal cavity results in death in 1 to 3 days. Death is apparently due to cholemia since the peritoneal bile is often found sterile. Many cases of traumatic rupture of the biliary tract in the human have resulted in death, but with less rapidity than in the experimental animal. Death is probably the result of the absorption of bile salts which in the dog act more rapidly because of a greater percentage of toxic taurocholic acid.

Is there any danger at operation in peritoneal soiling with concentrated bile from the gall bladder in chronic gall bladder disease? There is danger from two possible sources—infection and bile salts. Death may be the result of either if sufficiently extensive in a patient with poor resistance. Another factor of great importance as far as morbidity is concerned is the formation of extensive disabling adhesions which are the result of careless surgery with its needless trauma, spilling of bile and spreading of infection. Any surgeon who has removed a few gall bladders after previous drainage must have been impressed by the frequently extensive adhesions and wondered how so much peritoneal irritation could have been produced by simple cholecystostomy. Such patients have undoubtedly in many cases been made worse by operation. Their gall bladder disease has not been cured and the pathology has been increased. Such results warrant our most conscientious consideration. In any operation on the gall bladder or gall ducts great care should be taken not to permit bile to escape into the abdomen. If cholecystostomy is to be done the gall bladder should be thoroughly emptied with a trocar before making the incision for a drainage tube. If there is no history of common duct obstruction with fever, chill and jaundice, it is rarely necessary to open either the gall bladder or common duct when doing cholecystectomy unless the latter is found dilated.

Cholecystectomy for disease of the gall tract does not result in 100 per cent cures. This is quite to be expected when extensive pathologic changes exist in the liver and perhaps in the pancreas, with adhesions involving the duodenum, pylorus and transverse colon as a result of advanced disease, acute infection or multiple operations. Johnson and Pearre (Southern Medical Journal, Dec. 1925) report 60 per cent of their patients cured and 37.5 per cent improved after cholecystectomy. With cholecystostomy they cured 51 per cent and improved 42 per cent. While these results probably represent the average obtained in all cases by most surgeons, any such statistics should be carefully analysed before definite conclusions are drawn. In our hands choleystectomy as a secondary operation has proven less satisfactory from a curative standpoint than has this operation when done primarily. To properly estimate the value of this operative procedure it should be judged without the handicap of a previous operation making cure more difficult.

Good results with cholecystectomy will depend to a great extent upon early treatment. Diagnoses are now being made earlier in the disease and I feel sure that our operative cures will be greater in the future.

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Recent Advances in the Treatment of Pernicious Anemia

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Every clinician realizes how unsatisfactory has been the treatment of pernicious anemia. Many different drugs and methods of treatment have been suggested. Hydrochloric acid in large doses, arsenic and transfusion have stood the test of time as therapeutic measures of proven value. Conner[1] has recently reported promising results from the oral administration of gentian violet and acrivolet. The measures employed have however, only aided in initiating or prolonging remissions. They have not materially altered the inevitably fatal course of the disease.

Most students of pernicious anemia agree that the disease must be of gastro-intestinal origin. Numerous workers beginning with Herter have thought that poisons having an affinity for the hematopoietic and nervous systems may be due to the predominance of B. Welchii in the intestinal flora. The proof offered for this view is not convincing. The same may be said for the

theory that certain types of yeasts may be the causative agent. The absence of free hydrochloric acid is a constant finding in pernicious anemia. It is most probable that digestion in the absence of free acid is constantly abnormal, especially with reference to the splitting of proteins and fats. It is quite possible that such is the source of the toxic bodies in this disease.

Clinicians have emphasized that patients suffering from pernicious anemia have often taken for a long time an incomplete diet. Bohan[2] has especially called attention to the fact that there is commonly a protein deficiency over a long period. Patients sometimes give a history of taking an excess of fats.

Koessler, Mauer and Laughlin[3] think that changes in the viability and permeability of the intestinal wall may be important factors. Certain products of normal digestion are toxic if introduced into the blood stream. Normally the intestinal wall is an effectual barrier to the entrance of poisons into the blood. McCarrison has pointed out the very striking changes which take place in the intestinal wall as the result of vitamin deficiency. Koessler, Mauer and Laughlin produced a marked anemia in the rat by feeding a vitamin deficient diet, which they think is due to altered permeability of the intestinal wall. They state that suggestive good results have been obtained in patients with pernicious anemia by feeding a vitamin rich diet.

Certainly the most promising results so far obtained from any method of treatment of pernicious anemia are those reported recently by Minot and Murphy[4]. They have treated a large series of patients with a full protein diet including at least a half pound of liver a day. Fats have been much restricted, and fruit and vegetables supplied in abundance but liver is seemingly the most important item in the diet. The use of liver is thoroughly justified from experimental work since Whipple and Robscheit-Robbins[5] found that it is especially valuable in hastening blood regeneration in dogs with experimental anemia.

It is as yet not clear whether the favorable action is due to the fact that liver is a complete protein or contains some specific antihemolytic factor. Conclusions must certainly be cautiously drawn since pernicious anemia often runs a bizarre course with frequent spontaneous remissions. Clinicians elswhere are, however, experiencing equally good results as those reported by Minot and Murphy by the proper use of the diet suggested by the Boston investigators. There is uniformly a rise in the reticulocytes and a fall in the icterus index indicating there is more active regeneration and less active destruction of red cells with the liver diet.

In Table 1 is shown the diet recommended by Minot and Murphy[6]. It is essentially a high protein diet, containing about 200 grams of mammalian liver each day, rich in fruit and vegetables, sharply limited in fats and including only a small amount of milk.

I have had excellent results with this diet in all cases of pernicious anemia in which it has been used.

The following case history illustrates well the effect of the diet in contrast with other therapeutic measures.

CASE HISTORY

M. B., a housewife, age 40, was first seen in May, 1925, complaining of weakness, shortness of breath and nausea. She had had no serious disease before the onset of her present illness. She stated that she had not felt well for three years. For several months she had had increasing gastro-intestinal disturbances, especially belching and distention, weakness and distention. Before admission to the hospital she had had sore tongue and mouth, tingling of the hands and feet and had lost 20 pounds in weight. She had been habitually constipated. There had been no attacks of diarrhea.

The general physical examination showed little except the general evidence of anemia. There was a definite chronic infection of the tonsils, and a chronic right maxillary sinusitis.

The blood examination on admission showed: red blood cells, 1,830,000, hemoglobin 48 per cent, color index, 1.41, volume index, 1.58, white blood cells, 6,600, stained film, poikilocytosis, and anisocytosis without nucleated cells. The gastric analysis showed no free hydrochloric acid, with a total acidity of 8 acidity per cent. The urine was negative.

With rest in bed, forced feeding, and dilute hydrochloric acid she began to improve. During her stay in the hospital four transfusions of 500 cc, each were done. On discharge five weeks later the blood examination showed: red blood cells, 3,450,000, hemoglobin 79 per cent, and white blood cells 4000. One month later Dr. Sam Roberts removed the tonsils and cleaned out the right maxillary sinus.

She was seen at intervals of six or eight

weeks. With hydrochloric acid, Fowler's solution, and regulation of the diet, her condition remained satisfactory for a number of months. The highest red count during this period was 3,264,000, and hemoglobin 78 per cent. In March, 1926, her count began to fall and her symptoms to return in spite of treatment.

On October 5th, 1926 she was again admitted to the hospital with a red count of 1,056,000, hemoglobin of 50 per cent and a white count of 6,500. At this time the diet of Minot and Murphy was begun. Her treatment otherwise was the same she had had for the previous 18 months. She showed a rapid improvement as indicated in the following blood count.

	Red blood count	Hemoglobin %	Packed cells %
Oct. 5, 1926	1,056,000	47	50
Oct. 15, 1926	2,064,000	62	65
Oct. 23, 1926	3,096,000	73	75
Oct. 30, 1926	3,200,000	74	77
Nov. 11, 1926	4,024,000	91	96

She has kept up the diet and her blood count when last seen was still high. She had a more rapid improvement and better remission with this diet, than with the other method of treatment. It seems reasonable to suppose that diet was responsible for the rapid improvement.

This patient illustrates well the general principles of management of pernicious anemia. In an active stage rest in bed is the most important factor. Hydrochloric acid should be given in large doses. I begin with ten drops of the dilute solution and increased rapidly up to 90 drops, three times a day, with the meals. Arsenic in some form might be given although I am inclined to believe that the value of arsenic in pernicious anemia has been much overestimated. The bowels should be kept freely moving. Infection such as this patient presented should be removed. This is done because the infection is an added load for the patient to carry, not because it is the etiologic factor. Transfusions are of value when properly used with extreme anemia. A transfusion may be life saving in tiding a patient over until a remission begins. When a remission is already under way transfusions often hasten the improvement and shorten the stay in the hospital. The results from transfusion when the patient is on the downgrade are very disappointing. The most hopeful addition to the treatment of pernicious anemia is the diet introduced by Minot and Murphy. Only time will tell what its ultimate value will be.

TABLE 1

FOODS FOR PATIENTS WITH PERNICIOUS ANEMIA

Mammalian Liver Essential: About 200 grams (cooked weight) daily.

1 Encourage (in order of importance)—
Mammalian liver, calves, beef, pig from 90 to 240 gms. daily.
Permit—one egg, milk (240 gms.) tea ad lib., coffee ad lib.
Avoid fats not to exceed 70 gms. per day, butter.

2 Encourage kidneys.
Permit—Cocoa shells ad lib.
Avoid—Cream, cheese.

3 Encourage chicken livers.
Permit—Potatoes.
Avoid—Fat of meats, bacon.

4 Encourage red muscle meat (trimmed of fat)
(a) Beef.
(b) Lamb mutton. .
(c) Calves' heart, beef heart.
Permit — Macaroni, cereals, toasted whole wheat bread, simple crackers.
Avoid—Pork, oils, cotton seed oil, olive in dressings, etc.

5 Encourage fruits (fresh, canned or dried)
(a) Peaches.
(b) Apricots.
(c) Oranges.
(d) Strawberries.
(e) Grapefruit.
(f) Pineapple. .
(g) Blueberries..
(h) Raisins (steam until soft before serving).
(i) Prunes.
(j) Other fresh fruit. About 400 gms. per day.
Permit—Sugar not to exceed 20 gms., per day, pickles, relishes, salt and vinegar, poultry, fish, shell fish.
Avoid—Nuts.

6 Encourage Vegetables (cooked or raw).
(a) Lettuce.
(b) Spinach.
(c) Asparagus.
(d) Cabbage.
(e) Tomatoes.
(f) Other vegetables containing from 1 to 10 per cent of carbohydrate.

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Obesity

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Obesity is a *symptom.* Because it is associated with a variety of conditions, the obese patient needs careful study before any attempt at reduction is begun. Otherwise, a body already suffering from lack of proper nourishment may be injured by dieting or a diseased heart injured by exercise.

The classification of obesity as exogenous or from food ingested and endogenous or from endocrine disturbances is not necessary because all fatty tissue is built up from material that originally entered the alimentary tract[1] [2].

With intelligent treatment as an objective, four classes of obese patients are recognized: [3] [4]. Obesity that is *inherited,* obesity that occurs with *endocrine disturbances* obesity associated with *cardiorenovascular* disease, and obesity that is directly *dependent upon foods ingested.* These types often overlap. A pure type is exceptional. For example obesity appearing at the menopause may be an herditary trait, and there develop simultaneously an increased appetite and myocardial degeneration. Usually, however, a predominating feature places each case in one or the other class, and this is helpful in treatment.

Hereditary obesity may be decreased slightly by diet and exercise, but actual benefit thus derived is doubtful. Inherited obeseness is as normal for certain individuals as is inherited stature. Turkish people and some African tribes are normally obese. . In securing a history searching for hereditary factors, not only must parents and grandparents be taken into consideration but also aunts, uncles, brothers, sisters and great grandparents because the Mendelian law holds good here as elsewhere and subdominant characters may be suppressed through several generations.

Fat is deposited *generally* or *locally.* It may be chiefly at the girdle, the neck, legs, or breasts. Inherited obeseness may appear only at a certain age. In some families, the babies are large at birth but grow into small individuals. Girdle obesity at the age of fifty is often a family trait.

The endocrine type of obesity may be inherited, but there are also individual cases[5] such as the eunuch, cretin, or subject of Froelich's disease. Obeseness often appears at certain periods of endocrine adjustment, at puberty and at the menopause, at marriage[6] and with pregnancy. Obesity frequently developes after ovariectomy.

The local fat deposits dfinitely associated with endocrine gland changes have been named according to clinical and pathological findings. There are adiposis dolorosa, adiposis tuberosa simplex, symetrical adenolipomatosis, adipositas cerebralis, adipositas pinealis, nodular circumscribed symetrical multiple lipomatosis, lipodystrophia progressiva[7] and adiposis secondary to neuropathic edema[8]. In cases of true Dercum's disease, definite anatomic changes have been found in the thyroid, pituitary and sex glands[9].

Cases of thyroid deficiency respond notably well to thyroid medication. In ovarian deficiency, extracts of the sex glands given hypodermically or by mouth occasionally lead to improvement. I have never seen obeseness of the pituitary type described by Engelbach[10] reduced by hypophyseal medication in any form. [11] [12] [13] It has been pointed out that in many cases of so-called pituitary obesity there is no anatomical alteration of the hypophysis, but changes in the adjacent brain tissue have frequently occurred[14] [15]. I believe that the favorable reductions described in so-called pituitary obesity were due not to the hypophysis administered, but to other constituents of the mixed glandular material, especially thyroid.

In obeseness of long duration, regardless of type, there are associated heart, kidney, blood and vessel changes. It is difficult to determine whether the obeseness causes vascular change or the vascular alterations determine the obesity—or whether both are dependent upon a third factor. But the intimate association of obeseness with heart, kidney and vessel disease is well known.

The metabolic disturbances, diabetes, uremia and gout[16] [17] [18] obscure conditions reluctantly considered disease entities, and notoriously associated with heart, kidney and vascular changes, are frequently accompanied by obeseness. Such conditions should be sought before a cure for the symptom obesity is outlined. Diabetes with its dirth of characteristic objective symptoms, or incipient retention of nitrogenous elements may be easily overlooked when the obese patient is examined. The fat diabetic

requires different management from the fat nephritic[19].

Having determined in a given case that the symptom obeseness is associated with diabetes, further search for the basic cause is indicated. It may be syphilis. Where in another case obeseness, vascular change and hypertension are present, the prime etiologic factor may be lead or some other poison.

Obeseness occurs with chronic anemia. Scrupulous search may place the anemic condition in the secondary instead of the primary group.

Obesity due chiefly to feeding conditions constitutes the smallest group, yet the chief treatmens for all cases is usually limitation of diet[20 21]. However, many obese individuals not only remain fat but actually gain weight on a diet scientifically calculated to be less than the minimum basal requirement. They starve, grow weak, and gain weight. On the other hand, certain slender individuals when stuffed full of food weigh less than when fed only a little above the basal requirment. Superalimentation does not necessarily lead to supernutrition[22]. This is recognized by many men in the treatment of tuberculosis.

In a few instances, dietetic indiscretions lead to obesity. The artirficially fed infant is often pudgy fat. Obeseness may follow years of beer drinking. An obese condition associated with anemia in young people is improved by the addition of iron to the diet. Obeseness has been described associated with chronic constipation reduced by a nonconstipating diet.

SUMMARY

Since obesity is a symptom, the underlying cause should be sought before treatment is undertaken. The treatment of four arbitrarily chosen groups of obese cases is dicussed.

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Perverted Physiology as a Factor in Surgical Diseases of the Biliary Tract.

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Biologically, the liver is the second oldest organ in the body, the gut tract being the oldest, and the liver formed from the first off-budding from the foregut. It is the largest organ in the body, weighing approximately fifty ounces in the adult male. It is within itself a complete biochemic laboratory, situated between the portal and systemic circulation. It is both an organ of secretion and excretion.

Hess has attempted to ascribe to certain sections of the liver certain specific functions. Silvestri, likewise, has attempted to localize within the liver certain specific functions. He called attention to the fact that in Banti's disease, which is primarily a splenic affair, that it is the left lobe of the liver that enlarges; while in diabetes, which is pancreatic in origin, it is the right that shows most marked changes.

It is interesting to note that all of the digestive enzymes are secreted by off-buddings from the foregut; that is, that the entire function of digestion is controlled by the foregut, while the midgut forms the area from which absorption takes place.

The liver has an unusual vascular supply, an arrangement of afferent and efferent

blood vessels that is found in no other organ of the body. While other organs do their work in the presence of oxygenated blood, the liver works with and in the presence of blood that is entirely of a venous type. If one compares the volume of blood entering the liver from the portal circulation with the relatively small amount that enters from the hepatic artery, one is impressed with the insignificance of the role played by arterial blood. In fact, some anatomists insist that the branches of the hepatic artery do not reach to the liver cells proper, but merely supply the supporting structures of the liver and its blood vessels.

The liver has, also, an unusual cellular arrangement or cellular architecture. The individual cells are of the simplest form of cell life found in the body. They are incapable of work hypertrophy, but possess to a superlative degree the power of regeneration.

Mann removed seventy per cent of the liver in a dog and demonstrated that in a comparatively short time the remaining thirty per cent regenerated to such an extent that the dog's liver was soon of its original size.

Rous and McMaster ligated ninety per cent of the bile ducts without jaundice occurring. The ligated portions of the liver underwent atrophy with marked and rapid contraction; while the remaining ten per cent regenerated to such an extent that the functional part of the liver was within three months equal in volume to the original liver. The cellular reaction to injury or insult is death, with replacement by fibrous tissue. While the remaining cells rapidly reproduce new ones to take the place of the destroyed liver cells, yet the destruction is marked in each instance by an increase in the amount of connective tissue.

The function of the liver is both secretory and excretory, and while our physiologists are not yet certain as to the details of the functions of the liver, there are certain functions, however, that may be safely predicted.

The liver is the great reservoir for converted sugar in the form of glycogen. Closely allied with the glycogen function of the liver, there is an undoubted reciprocal relationship with the pancreas. The liver, likewise, is active in the metabolism of proteins and the catabolism of purins; it aids in the maintenance of the ammonia balance; it has to do with the production of urea. Another function that ranges high, possibly first, is the diaminization of the amino acids; it has to do with the storage and liberation of fats; the clotting of the blood is in some way controlled or influenced by the liver. Then again, there is the more obvious function of the liver; that is, the secretion and excretion of the bile. It possesses a marked detoxifying power whereby noxious by-products from the gastro-intestinal tract or from bacteria are rendered inert or harmless.

The question naturally arises: By what mechanism is the liver injured? This is accomplished, not only by the injurious by-products from the intestinal tract, but by both bacteria and toxins arising from the death and breaking up of the bacteria, which are being brought to the liver; both by the portal circulation and by the abdominal lymphatics. The live bacteria may produce capillary infarcts, resulting in the destruction of the life of the bacteria. The liberated toxins permeate well beyond the walls of the capillaries. The effect is death of the liver cell with replacemet by fibrous tissue, and as one views the increase in fibrous tissue in the liver we see constantly associated with gall bladder disease, one is impressed with the fact that this fibrous tissue represents the summation of millions of minor insults to which this liver has been subjected day after day and year after year for a long period of time; and that, likewise, the infected gall bladder is an expression of a pathological process much more wide spread than is apparent in the gall bladder itself.

These insults in the form of bacteria or bacterial toxins have several routes of entrance into the liver: The arterial system, which may bring infectious products from distant portions of the body is probably of minor importance; direct extension from infected contiguous organs; and possible ascension through the bile ducts are, likewise, of infrequent occurrence. We are impressed that the two chief routes of insult are through the avenue of the abdominal lymphatics or the portal circulation.

The effect of abdominal infections upon the liver has been emphasized by Graham, he declaring that there is no such thing as gall bladder disease without hepatitis and cholangitis. He even suggests the possibility that hepatitis may be the primary lesion; that the infection of the gall bladder is secondary, and accomplished by means of extension from the liver through the lymphatics.

Heyd removed sections of the liver for

purposes of study in the cases of operations done for the removal of the appendix, carcinoma of the stomach, etc., as well as infected gall bladders; and he, likewise, agrees with Graham that any long continued abdominal infection is bound to record its effect upon the liver; and both Graham and Heyd have called attention to the fact that in the presence of an infected gall bladder, the increased amount of fibrous tissue in the liver is more marked adjacent to the gall bladder; in fact, the amount of fibrous tissue decreases as the distance increases from the gall bladder.

Physiological chemistry has demonstrated that the functions of the liver are manifold. It is only partially an excretory organ, its main function being concerned with syntheses and desyntheses for the blood. Probably the most important role played by the liver in the physical economy of the blood is the diaminization of the amino acids and the synthesis of urea. Many methods have been tried for the determining of the impaired physiological function of the diseased liver. Most of these have yielded indifferent results. The determining of non-protein nitrogen is of some value. Many show a definite increase in blood sugar, the blood sugar returning to normal after the removal of a diseased gall bladder; on the other hand, some show a hypoglycemia. Some have tried to gain information by determining the cholesterol content of the blood. This is, however, of questionable value. One fact has been established, and that is that an increased cholesterol content is constantly present in obstructive jaundice, regardless of the cause of the jaundice; likewise, the determination of fibrinogen has yielded little information of value; in fact, it seems probable that the amount and the character of the calcium is of more importance in determining clotting time than is fibrinogen in the jaundiced individual.

The carbon dioxide combining power is of definite importance. It finds its chief value in determining whether the patient is approaching an acidosis or alkalosis. In the presence of a definitely reduced liver function, the condition of alkalosis is one to be feared, and one ought never to use alkalies promiscuously in gall bladder disease, without first having determined the carbon dioxide combining power.

A consideration of the physiology and biochemistry leads one to a much broader view concerning symptomatology of gall bladder disease. This symptomatology must of necessity include metabolic and systemic disturbances that are directly due to impairment of liver functions. It leads to a recognition of the fact that there are in addition to the local symptoms, a group which can be termed remote symptoms, and a third group of symptoms, systemic in character.

The first group or the local symptoms are the one generally considered, and with which we are all familiar.

As examples of the second or remote group, we would suggest arthritis, nephritis, arterio-sclerosis, endocarditis, myocarditis, and even a group of symptoms simulating angina pectoris.

The systemic or general symptoms which can be explained by this broader conception of the physiology of the liver are an ill defined group, represented by asthenia, loss of weight, a perverted skin pigmentation, and a general instability of the patient.

The post operative complications, likewise, take on an increased significance when one considers the perverted physiology present in biliary tract infections.

Many of the post operative complications readily recognized in the past are clear cut, distinct, and possess within certain limits a chronological sequence.

In the first twenty-four hours, the complications are anatomical; such as hemorrhage, shock, embolism, etc. In the succeeding forty-eight to seventy-two hours, the element of infection may possibly come into play, with the production of peritonitis and still later abscess formation.

We desire especially to emphasize the importance of the work of Heyd, Moynihan, and others who have called attention to clinical states that supervene after operations upon the biliary tract. Although these clinical complications are comparatively rare, yet they are definite, and apparently within their group are rather characteristic in their symptomatology. For convenience of description, they may be classed into three more or less distinct groups:

In the first, the patients usually have had comparatively simple gall bladder operations. They are usually obese, with a history of chronic gall bladder disease extending over a long period of time. The preliminary study of their urine, blood, etc, has assured us as to the competency of the renal function. Following the operation, the recovery from the anesthetic is delayed. They early develop delirium, which finally passes into coma. Many have been in the habit of attributing this condition to the

development of a post operative acidosis. In making a complete study of these patients, both before and after operation, Dr. Killian was able to show that the carbon dioxide combining power of these patients was far in excess of the normal, and approached values of seventy to eighty, sometimes even ninety volumes per cent; and that the condition, chemically, is that of an alkalosis.

The second group is essentially different in its clinical manifestations and comes on at a slightly later time. Usually the patient has had a very severe type of biliary infection, usually a history of jaundice, or a previous gall bladder operation. Their post operative progress has been satisfactory until the end of thirty-six to forty-eight hours, when they become irritable, and soon pass into a pronounced vaso-motor collapse. The condition may be associated with gastric dilation. There is usually ample renal function. The intravenous administration of glucose and saline has usually brought about a recovery. These cases do not exhibit the alkalinization of the preceding group, and their blood chemistry is only changed in so far as there is a change in the percentage of the rest nitrogen to urea nitrogen. These cases are regarded as due to the liberation of some pancreatic toxin with inadequate liver protection.

The third group presents a still more difficult phenomena. The patient has usually been progressing favorably up to the end of the fifth or sixth day. The preliminary studies have shown the blood chemistry, coagulation time; all, within the normal limits. Usually these patients have been jaundiced. At the end of the sixth to the tenth post operative day, with a constantly diminishing jaundice, these patients become sleepy, stuporous, and pass into a profound state of coma and die irrespective of any remedial agents employed. The clinical picture presented is essentially a coma occurring in a patient with a diminishing obstructive jaundice. In its manifestations, it is not unlike the terminal stage of a portal cirrhosis, with a complete liver exhaustion.

Heyd says, "We have long felt that these three clinical conditions were in some way connected with an impaired liver function; either a disturbed liver metabolism, a liver disfunction, or a liver insufficiency."

In the first type, the rapidity with which the development of coma is brought about takes it entirely out of the infectious class. The whole mechanism suggests a complete and rapid cessation of liver function.

The second type, which Moynihan has described under the caption "Pancreatic Asthenia," occurs too late for primary operative shock; and may be interpreted as being due to an inundation of the system with operative and infectious byproducts plus pancreatic enzymes, and direct poisoning of the liver cells with cessation of their function.

In the third group, it would seem probable that these patients have had sufficient liver function to maintain a certain degree of body wellfare and activity. When this is interrupted by a surgical operation, the liver is unable to withstand the additional load, and the interim between operation and death represents the period of increasing and progressive exhaustion of liver capacity.

It is our desire to emphasize the fact that, as one gazes upon an infected gall bladder, the condition of that gall bladder is an expression of pathological processes much more wide spread than is apparent in the gall bladder alone; and that as one notices the liver, each minute increase in fibrous tissue represents the reaction of that liver to an insult; that these insults have occurred in repeated showers over a period of years; that the rounded edge of the liver and the increase in connective tissue represent a summation of minute insults to the liver tissue, extending over a period of years; and what is more, it is evident that the incriminating bacteria gain their entry from various points and through various portals.

Let us urge a broader and more comprehensive study of these patients, with a full appreciation of the physiological and biochemic role of this most important organ, the liver.

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Diuretics

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The normal secretion of the urine is maintained by four factors:-the general blood pressure, the local blood pressure in the kidneys, the constitution of the blood and the specific secretory activity of the renal cells. These four factors are all constantly at work in the normal kidney and their activities are so closely interwoven that the normal passage of urine becomes a very highly complex process. In disease, when the patient excretes an insufficient amount of urine, we may find one or all or any combination

of the four at fault, and when we use diuretics in an attempt to promote a copious secretion of urine, we find the situation again exceedingly complex, since many diuretics produce their effect not because of one single action but because they influence simultaneously several of the factors that control the secretion of urine.

The rival theories of Ludwig and Heidenhain have now been the subject of physiological debate for more than sixty years. Ludwig, it will be recalled, saw in the secretion of urine a mechanical process produced by a filtration through the glomerulus, while Heidenhain, with perhaps a more sentimental regard for the kidney, saw in the secretion of urine a more vital process, a specific secretory activity of the kidney cells. Cushny, after a lapse of more than fifty years, reviewed the process in the light of his own extensive observations and of the enormous literature which had accumulated, and proposed the "modern theory" of urinary secretion. Cushny's theory in brief is, that the glomeruli of the kidneys act mainly as filters of urine, while the kidney tubules reabsorb certain substances in the urine and thus bring it to the proper concentration.

Cushny's theory, while reconciling to a certain extent the "mechanical" or filtration theory with the "vital" or secretory theory, stresses the importance of the mechanics of urinary production rather than the secretory function. In the study of those victims of insufficient urinary output who are in need of diuretics, we are usually impressed by the evidence of severe mechanical disturbances. Most of our patients develop edema because the circulation through the kidneys is at fault and the constitution of the blood is imperfect, or the filter itself has been destroyed.

One of the commonest causes of insufficient urinary output is cardiac disease. Cardiac insufficiency may be valvular in origin or may be the result of a long standing hypertension. In either event, the root of the trouble is myocardial and the myocardial insufficiency is so marked that the normal blood pressure, particularly the circulation through the kidneys, is not maintained. This condition indicates the use of a drug to increase the force of the heartbeat and to maintain the circulation through the kidneys.

Digitalis meets both of these indications. This most valuable of all cardiac drugs not only improves the tone of the heart muscle but also improves the circulation through the kidneys. Although in health, as Loewi and Ionescu have shown, it hastens the rate of the kidney circulation by producing a local vasodilatation of the renal vessels, there is no good evidence that this occurs with the dosage usually employed therapeutically. It is, as Eggleston states, primarily a cardiac diuretic. While digitalis may raise the blood pressure when it has fallen to a dangerously low level, the presence of high blood pressure is no contraindication to its use. Price showed in 1912 that digitalis produces no rise in the blood pressure of patients with hypertension, an observation which has been repeatedly confirmed. Vaquez has recently written that, "the fear of raising the arterial pressure by the administration of digitalis is illusory; the drug can be given without danger to hypertensive subjects as well as those whose pressure is normal or low."

A great many new preparations of digitalis have appeared on the market-most of them quite potent and effective, but, as Sir James Mackenzie remarks, "I have never found the tincture fail me, and it is this form which I have used most frequently, because it is the cheapest, and therefore in the reach of the poorest." The dosage of the tincture employed by Mackenzie was one dram per day given in three doses and continued until therapeutic or mild toxic effects are noted. Eggleston found that the full therapeutic effect of a standardized tincture was produced by 15 cc. (approximately 4 drams) per 100 lbs. of body weight. This method consists of giving one half this dose (2 drams) at one dose, following in six hours by one dram, and then smaller fractions every 4th to 6th hour until the total dosage or full response is reached.

Another valuable cardiac diuretic is strophanthin. This drug, Vaquez considers, "the heart medicine par excellence, the effects of which are exerted exclusively upon the myocardium and on the muscle fibers, whose tonicity increases without the intervention of other factors." Ouabain, one of the active principles of strophanthin, is perhaps the preparation of choice, since it can be crystalized and the dosage accurately standardized. The dose usually employed is 1/240 grain (one fourth milligram) repeated three or four times at intervals of twelve to twenty-four hours and injected intravenously.

In using strophanthin, we must not forget that it is very toxic. It was used for countless generations by the savages of Africa as an arrow poison before its therapeutic

value was recognized. It must be employed with great caution in patients who are taking digitalis.

Among the drugs usually classed as renal diuretics, the members of the caffeine series have attracted the most attention and have been the subject of most investigation. While the action of this group of diuretics was long explained on the theory of stimulation of the renal epithelium, the action is now considered to be mainly due to an improved circulation in the kidney produced by dilatation of the renal vessels. These effects, while especially marked in the kidney, also occur elsewhere and careful experiments have shown that hypodermic doses produce an increased systolic output and a definite increase of the total blood flow. No one who has systematically and persistently used such preparations as caffeine sodium benzoate in cardiac disease can doubt its effectiveness in increasing the tonicity of the heart, as well as in improving diuresis.

The member of the caffeine group of diuretics which is most generally used is diuretin or theobromine sodium salicylate. While this, with theocin or euphyllin appear to give good results in the treatment of edema, particularly of cardiac origin, the results obtained in patients suffering from acute or chronic nephritis have, in the main, been disappointing. The experimental work of Christian suggests that their use in acute nephritis may actually be harmful and, as Frothingham remarks in discussing the treatment of chronic nephritis, "it seems illogical to recommend drugs which have an effect upon the kidneys when all the remainder of the treatment consists in endeavoring to spare the kidneys from work." He adds that, "unfortunately, the various diuretics that work so well in cases of chronic passive congestion of the kidneys, such as preparations of caffeine or theocin, are usually of little benefit in chronic nephritis."

A very important group of diuretics are those that act by lessening the viscosity of the blood, thereby increasing its filtrability and raising the glomerular pressure. This very important mechanism is closely connected with the well known phenomenon of osmotic pressure, the role of which in diuresis has been especially studied since the work of Widal in 1903. Widal found that in edema the addition of a large amount of sodium chloride to the diet increased the edema, while a diet containing a very small amount of salt caused disappearance of the edema. From these results, Widal concluded that edema was caused by the retention of sodium chloride which, in time, led to a retention of water. This work led to the widespread use of the so-called salt-free—more properly salt-poor diets, which have been of such great value in the treatment of nephritic edema.

Water, which was praised so much by older clinicians as the greatest of all diuretics, acts by producing osmotic changes. Water, however, which has such a marked diuretic effect in health or in mild cases of nephritis, cannot be used in large quantities with patients having cardiac decompensation and is ineffectual in patients with any marked degree of kidney involvement.

Glucose, a member of this group, has a marked diuretic effect. A very striking illustration of its action is seen in diabetes mellitus where the patient usually voids large quantities of urine when he has a high blood sugar and large quantities of glucose in the urine. When, however, his blood sugar becomes normal and sugar disappears from the urine, diuresis ceases and his urinary output becomes normal.

In diabetes mellitus, the high sugar concentration of the blood draws water from the tissues and not only increases the urinary output, but may actually produce a dessication of the tissues. A similar series of events follows the introduction of a concentrated solution of glucose into the blood stream. This same principle is universally employed by neurological surgeons who reduce edema of the brain by the intravenous injections of concentrated glucose solutions.

We have had several gratifying results in acute nephritis with the intravenous use of glucose and have also found it effective in certain cases of chronic nephritis. We have usually employed 300 to 500 cc. of a 25% solution injected intravenously at intervals of three to four hours. Occasionally a patient has a chill and an elevation of temperature after the injection but such complications have been unusual and have not apparently been followed by any ill effects.

The saline diuretics apparently act in a manner similar to glucose, although their exact mechanism may differ somewhat. Schultz in 1917, while treating cases of war nephritis with the German army, found that calcium chloride produced striking diuresis in many resistant cases of edema. At the Mayo Clinic, Keith substituted ammonium chloride for calcium chloride and reported many striking results, some patients losing 70 pounds of fluid in five to six

weeks. Keith used ammonium chloride by mouth in doses from 200 to 400 grams over a period of two to four weeks. Our practice has been to give patients from 100 to 150 grains per day for a week or two.

One of the newer diuretics which has attracted much attention is novasurol or "merbaphen" as it is called in this country. Merbaphen is an organic mercury preparation first prepared for the treatment of syphilis and later, accidentally, found to have marked diuretic properties. The mode of action is still a bit uncertain, but it apparently consists in drawing both salt and water out of the tissues and into the blood, with a resultant polyuria.

This diuretic usually begins to act in three or four hours and the results are often striking. We recently had under observation a patient with nephritis whose urinary output was only 300 cc. per day. Following one subcutaneous injection of 1 cc. of merbaphen the urinary output was increased to 5000 cc. in a day. Merbaphen, however, should be employed with caution. The initial dose should be only 0.5 cc. and larger doses should not be employed oftener than once in four or five days. We have occasionally seen a watery diarrhea with blood and mucus in the stools following its use, but have not observed any serious complications.

Keith has employed a combination of ammonium chloride and merbaphen and has reported good results in many patients,—a finding which we can confirm. Both of these drugs used in combination are oftener much more effective than either employed alone.

The action of diuretics can often be assisted by other therapuetic measures. Saline cathartics are useful in promoting watery evacuations which diminish the total fluid content of the body and thus lessen the load which is thrown upon the kidneys. Another accessory form of treatment which is often of great value is incision or multiple superficial punctures of an edematous extremity. The amount of fluid which drains from an edematous leg following small superficial incisions or punctures is often very striking. Recently we studied the amount of fluid lost from an edematous pair of legs following multiple punctures with a hypodermic needle and found each leg to drain 250 cc. of fluid per hour. Such incisions or punctures should be made under aseptic conditions and care must be taken to prevent infection.

In the use of diuretics, the same principles must be observed as in all therapeutic measures. The cause of the edema should be first carefully studied and the appropriate diuretic employed.

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Meckel's Diverticulum

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Diverticuli, as applied to the digestive tract, are non-neoplastic outpouchings of the walls of the digestive tract having a lumen which may or may not connect with the lumen of the part from which they arise. They may be single or multiple and may be found in any part of the tract from the esopahgus to and including the rectum.

They are classified as true and false. Those having all of the coats of the organ from which they arise are true while those in which one or usually two of the coats are absent are false. They are also classified as congenital and acquired and even though Erdmann[1] and a few others believe that diverticuli of the colon are congenital, generally speaking the term congenital diverticulum refers to Meckel's diverticulum.

Meckel's diverticulum is said to be present in from one to two per cent of all individuals but lesions produced by it are found so infrequently in the course of any one medical man's practice that unless attention is occasionally called to it the average physician may not think of it when it is the offending part. Furthermore its symptoms are frequently so abrupt and so serious that it becomes an emergency case and immediate action must be taken and there is little time for investigations. The treatment of its lesions is always surgical, often radical and urgent major surgery. With the exception of umbilical fistulae, the conditions arising from it are never positively diagnosed and frequently not even suspected, even by the astute, until the abdomen has been opened. How often an acute abdomen is operated and the patient dies as a result of some condition arising from Meckel's diverticulum without the true cause ever being known I do not know. I must confess that I recall a case in my early practice where an aged woman died of an intestinal obstruction and a band running over the ascending colon was found at autopsy, that I now believe was a Meckel's diverticulum but at that time was not recognized.

For the above reasons and especially since some of the lesions have recently been de-

scribed and may not be generally known, a review of the subject and a report of a recent case will not be amiss.

History: Freidrich Christopher[2] says it was first observed by Lavater in 1671 and next by Ruysch in 1701, but was accurately described by John Friedrich Meckel, whose name it bears in 1812. His father was a distinguished professor of anatomy and surgery and his grandfather was the one who described Meckel's ganglion.

Development: "In the beginning of foetal life the mid gut is connected to the yolk sac by a wide canal called the vitteline duct. This duct is accompanied by an artery and two veins which maintains the circulation between the yolk sac and the intestines. At the end of a few weeks (about the 8th week) the duct becomes functionless and under normal conditions disappears; the atrophy beginning at the distal end. The vessels at the same time disappear except the portion in the messentery which persists and eventually forms the superior messenteric artery and vein. In about two per cent atrophy is not complete and varying portions of the duct remain and it is this remnant of foetal life which is known as Meckel's diverticulum." (Wellington)[3].

If the duct persists thruout there remains a fistulous tract extending from the small intestine to the umbilicus which may discharge mucus or even intestinal contents.

If the distal end of the duct atrophies and the vessels, although functionless, persist a band extending from the diverticulum to the umbilicus remains. At times the distal end only of the vessels becomes obliterated and leaves a cord extending from the diverticulum. This cord may become attached to any portion of the aodominal wall or its contents and it is these bands that so frequently cause intestinal obstruction and volvulus.

Meckel's diverticuli vary in size from mere projections to eight or ten inches or more. Rollester reported one 36 inches in length and Moll (4) one 33½ inches long.

Its walls are the same as those of the intestine from which it arises and may contain Lieberkuhn's glands and Peyers patches. Recently a number of writers have reported cases in which the distal mucosa was of the gastric type having acid secreting glands while the proximal mucosa was of the intestinal type having alkaline secreting glands.

Its lumen is usually the same size as that of the attached gut but occasionally the outlet is narrow or may have a valve action or be entirely closed. In this last case the secretions from its mucus lining accumulate and produces a cyst. McGlannon[5] reports an irregular flattened cyst of the diverticulum, 15 inches in its longest diameter and 8 inches in its shortest diameter, whose walls had become sarcomatous.

Its attachment to the gut is usually opposite that of the messentery but at times it is said to be at right angles to or even in the folds of the messentery. Occasionally it has a messentery of its own.

Its shape is generally that of a tubular pouch but it may be spherical or irregular.

It is usually located within 8 or 10 inches of the ileocecal valve in infants and within 3 feet in adults but it may be anywhere along the ileum or jejunum and hence may be found anywhere in the abdominal cavity.

Frequency: It is generally stated to be found in from 1 to 2 per cent of all individuals but most reports I have seen show that it is found much less frequently.

Christopher quotes Doefner's and Tellings statistics as follows:

	Autopies	With Meckels	
Doefner's—			
At Zurich	1822	14	
By English and French	4848	90	
Total	6670	104	1.5%
Telling's—			
Dresden City	8133	8	
Johns Hopkins	2600	15	
Boston City	1382	11	
Bender Hygienic Lab.	953	5	
Total	13068	39	.3%

Balfour in 10600 consecutive laparotomies only found 15 Meckel's diverticulum (.14%) and of these in only 5 was it the cause for the operation.

McGlannon[5] only observed 3 cases in more than 14,000 intra-peritoneal operations and says that Coley and Fortunne say in 18,000 autopsies only 15 Meckel's diverticuli were found.

Conditions which may arise as a result of Meckel's diverticulum are:

1—Intestinal obstruction including volvulus and intussusception.
2—Tumors.
3—Umbilical fistula.
4—Contents of a hernia.
5—Diverticulitis.
6—Intestinal hemorrhage.

The first four of these I will discuss very briefly.

Intestinal Obstruction: It may readily be seen that the bands described above may easily produce an obstruction. If the distal end of the band is attached to the umbilicus the gut may through its peristaltic action loop around it somewhat like clothes on a clothes line in a hard wind. If the distal end of the band becomes attached to other portions of the abdominal wall or to other organs or the messentery the gut may also become twisted over it or the band may contract down over the gut producing an obstruction. Volvulus may occur as a result of the twisting of the intestine around the fixed point made by the band. Intussusception results in these cases where the distal end of the diverticulum is not attached and its mouth is large permitting the diverticulum to be inverted into the intestinal canal and then pulling a portion of the intestine along with it.

Halstead reported by J. R. Wellington[3] estimates that 6 per cent of all cases of obstruction are due to Meckel's diverticulum.

Umbilical Fistula: If no atrophy of the omphalomessenteric duct occurs there remains an open tube from the ileum to and through the umbilicus and when the cord is tied the opening does not heal and a fistula persists discharging either mucus or intestinal contents or both. Occasionally the intestines are herniated through the duct and are ligated along with the cord and the infant dies of intestinal obstruction.

Contents of a Hernia: Where no atrophy of the omphalomessenteric duct occurs and the clefts of the abdomen do not close tightly one can readily understand that an umbilical hernia may easily contain a Meckel's diverticulum and it is found there more frequently than in any other hernia, but on account of its variability in position in the abdomen and in size it is also easily seen that it may be contained in the sac of any abdominal hernia either alone or with omentum and intestines. As early as 1770, forty two years before Meckel described it, Littre observed a Meckel's diverticulum in a hernial sac.

Tumors: The formation of a tumor from Meckel's diverticulum results in 4 ways, viz:

1—If its mouth is large foreign bodies may enter.

2—If the mouth has a valve like action intestinal contents may enter but not leave the diverticulum.

3—If its mouth is closed the secretions of the mucous membrane may be retained.

4—As a result of inflammation alone or in conjunction with any of the other causes, it may become adherent to omentum or bowels and an inflammatory mass arise.

Diverticulitis: During peristalsis the contents of the bowels are moved forward and backward and if there be a rent in the wall of the intestine some of its contents will sooner or later be forced through the opening. This is practically what we have in diverticulitis: The diverticulum becomes filled with intestinal contents or perhaps foreign bodies that have been swallowed and an inflammation results in the same manner that Mr. David Wilkie explains the cause of appendicits. If the inflammation is mild there may be a few cramps which soon pass over. If more severe the pains will be accompanied by vomiting, fever, rigidity, tenderness and leucocytosis. If very severe gangrene and perhaps perforation results with peritonitis and all of the symptoms of a fulminating appendicitis. Indeed the symptoms are so like those of appendicitis that probably every case of diverticulitis has been diagnosed appendicitis.

Several writers have tried to give a differential diagnosis of the two conditions and while a diverticulitis may be suspected on account of the point of tenderness and rigidity being superior and medial to McBurney's point a positive diagnosis is impossible.

Fronticelli[11] in an article, June, 1926, entitled "Acute Diverticulitis" says that only about 80 cases have been reported.

Intestinal hemorrhage: As stated before, the distal mucosa may contain alkaline glands and at the junction of the two ulcerations may occur and produce hemorrhages. Like other peptic ulcers the hemorrhage may be severe or mild and there is usually a history of repeated attacks.

Some 18 or 20 of these cases have been reported mostly by continental writers. In 1924 L. Guibal[6] in a report of a case of peptic ulcer of Meckel's diverticulum causing profuse intestnal hemorrhages said "very few of these cases, only 5 or 6, have been reported and the greater number of them have perforated." He says the first case was reported in 1913 by Hubschman. The history of his own case was a boy 14 years old who had intestinal hemorrhages in July, 1923, lasting a month. In January, 1924, hemorrhage recurred. Resection of intestine with Meckel's diverticulum, the ulcer having perforated. The mucosa above the ulcer was of the gastric type while that below it was of the intestinal type and the

ulcer was situated at the junction of the two types.

The history is a representative history in these cases except most of them occur at an earlier age. Guibal also cites the cases of Graman in 1915. Mulengracht in 1918, Muller in 1919, Meyevand and Duvant in 1922 and Hollopeau and Humbert in 1924.

Stulz and Worringer[8] report two cases of their own and eleven in the literature, ages 5 months to 15 years, all boys. 10 of the 13 perforated, 7 died from perforation with peritonitis, 1 died of anemia with localized peritonitis and 5 were saved.

In this country Abt and Strauss[7], Chicago, have been the most prolific writers on this type of cases. They report 3 cases, a girl 2 years and 2 boys each 11 months old.

Reginald Jackson[9] Madison, Wis., in 1924 reported a boy 10 years old with history of repeated hemorrhages and cramps in left mid-abdominal region for years. Exploratory operation with resection of Meckel's diverticulum and recovery, but no report of pathological or histological findings.

W. J. Mayo[10] in Oct., 1926, reports a boy, 15 years old with a history of repeated intestinal hemorrhages since he was 3 years old in whom an appendectomy had been made. He resected an inflammatory Meckel's diverticulum but gives no report of the histology of the mucosa.

Where no histological report has been made as in these last two cases, most writers seem to accept the conclusion where there has been hemorrhages that that is proof of gastric mucosa.

George Schaetz (Abt & Strauss)[7] shows that gastric epithelium was found at the tip of the diverticulum in 5 out of 30 cases (16.6%) that were examined.

Guibal[6] in his article "Peptic Ulcer of Meckel's Diverticulum Causing Profuse Hemorrhage" makes the following conclusions:

1 All ulcers bleed and give rise to abundant hemorrhages.

2 The greater number perforate.

3 They always occur in young individuals and most frequently in boys. The youngest 11 months and the oldest 28 years.

4 The presence of gastric mucosa was found in nearly all cases.

5 The ulcer forms at the junction of the gastic with the intestinal mucosa.

6 The origin of the gastric mucosa is unknown and there has been no satisfactory explanation of its presence.

7 Treatment is the radical cure of the diverticulum by early operation.

8 The ulcer appears to be the result of contact of the gastric juice with the intestinal mucosa.

In the differential diagnosis of intestinal bleeding in children Abt & Strauss[7] say when an infant bleeds from the bowel intussusception is the condition first thought of, consequently a bleeding Meckel's diverticulum must be differentiated from this condition. The initial sharp pain and colic, as well as the vomiting are marked in intussusception and usually absent in the bleeding diverticulum. In intussusception the blood is usually small in quantity and intimately mixed with mucus. In the bleeding Meckel's diverticulum the blood is abundant, not mixed with mucus. In some instances it may be bright red. Frequently it is black and tarry. If a tumor is felt, the diagnosis of intussusception may be presumed, although the symptom is not always elicited.

"Rectal polypi and dysentery also produce bloody stools but their differentiation should cause no difficulty.

"Bleeding duodenal ulcer which occurs in children is usually ruled out by the roentgen rays, which shows a defect in the duodenum characteristic of the condition.

"If bleeding persists and a presumptive diagnosis of a Meckel's diverticulum can be established exploratory laparotomy is indicated."

Results of conditions arising from Meckel's Diverticulum: Wellington[3], in a study of 326 cases where diverticulum was the cause of the trouble but not including cases where it was found at autopsy or in the course of operations for other causes and was not involved, found the following conditions:

144 intestinal obstruction, not including intussusception and volvulus, with a mortality of 49%.
59 intussusception with mortality 54½%.
50 acute diverticulitis (in 15 the exciting cause was foreign bodies) mortality 40%.
27 included in hernial sac.
21 in which the diverticulum was open.
9 volvulus, mortality 50% 8 operated.
6 perforation in typhoid fever mortality 100%.
2 perforations from trauma.
3 prolapse of bowel.
1 pelvic tumor.
1 cyst.

This report was made in 1913 and an

analysis of more recent cases might show some improvement in the mortality on account of better technique in operating and earlier diagnosis, but nevertheless the mortality still remains very high and when a Meckel's diverticulum is found in the course of another operation, it should always be removed unless the condition of the patient is such that it entails too great a risk to do so. Miles F. Porter long ago said that the presence of a Meckel's diverticulum was a far greater menace to its possessor than is the appendix.

Treatment: I will not discuss this except to say that the only treatment of a Meckel's diverticulum and its complications is radical surgery.

Case—Hospital No. 3313-1926.

A. E. age 10, school boy entered St. Margaret Hospital Nov., 11, 1926 complaining of pain in his right upper quadrant. His history in brief was that three days prior he had abdominal cramps while at school and was sent home. The pain continued but there was no vomiting except after taking castor oil which his mother gave him the first day.

Past History: Negative except similar cramps three weeks before which were not so severe or persistent.

Examination: Temperature 100, pulse 96, tenderness and rigidity over the right lower quadrant and blood examination Hg. 90, W. B. C. 24,500, Polys. 88, L. L. 2, S. L. 10. The urine was normal containing no blood and no pus cells, and aside from the findings stated the examination otherwise was essentially negative.

Preoperative Diagnosis: Acute appendicitis.

Operation: Patient was immediately sent to the operating room and under ether anaesthesia a right rectus incision was made. Before opening the peritoneum a moderate amount of thin creamy pus escaped. Exploration for the appendix revealed a mass the size of a child's fist was palpated, which proved to be a gangrenous perforated Mechel's diverticulum. The diverticulum was about four inches long and 1¼ inches in diameter. Its base was attached to the ileum at right angles to the mesentery while it layed on and appeared to be a part of the mesentery. One half inch distal to the base was a round perforation the size of a goose quill and the whole mass looked like a very large gangrenous appendix.

About five inches of the ileum with a V shaped excision of the mesentery including the diverticulum was resected and lateral anastomosis made. A wick drain was inserted into the pelvis and another to the point of the anastomosis. 600 cc. of salt solution was given subcutaneously on the table and the patient made an uneventful recovery and at this time is perfectly well.

Pathological report: Drs. Wahl and Helwig made close research for gastric glands in the mucosa but failed to find them which was as expected as these have never been reported except in cases that have had ulceration and intestinal hemorrhages.

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Chronic Colitis

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In the treatment of chronic gastro-intestinal disorders, so much attention has been given to organs within the reach of surgical

influence that certain other organs have not received the consideration they deserve.

The internist has been so occupied with the newer problems of physiology, chemistry and metabolism that it may be said he has to an extent neglected the possibilities of gastro intestinal therapy.

There has crept into our medical teaching a certain degree of medical therapeutic nihilism, an attitude of mind that would relegate, in the final analysis, most cases of chronic dyspepsia or indigestion to surgery.

The skill and keenness of our surgeons coupled with the divided attention of our internists is largely responsible for this attitude. None will deny the good results obtained by the removal of a diseased gall bladder or appendix, but to adopt an attitude that practically all chronic gastro-intestinal complaints are caused by these organs is not conducive to better therapy.

Entirely too much attention has been given the gall bladder and appendix and too little to the colon as a cause of chronic gastro-intestinal symptoms.

For many years we believed in the existence of chronic appendicitis but happily most of the profession have come to the conclusion that there is no such thing as a chronic inflammation of the appendix which acts continuously and persistently over a period of months or years producing various forms of indigestion.

Yet many operations are still performed, apparently normal appendices removed, and paradoxically the patient is often freed from complaints following the operation.

It is recognized that a recurrent acute inflammation of the appendix occurs, the patient during each attack presenting the classical symptoms of an acute or sub-acute appendicitis but usually remaining symptom free in the intervals between attacks. This is not the type of case I refer to. I refer to the patient who has never had an attack of acute appendicitis but who has a chronic indigestion with a long history of pain in the abdomen often confined to the right side. None of the symptoms, such as fever, vomiting, right rectus rigidity or increased neutrophile count are present.

What then is the answer to the apparent enigma, a patient who has no disease of the appendix, yet is cured by an operation for its removal? For many years I studied this question. During the time my work was confined to x-ray I had an opportunity to study many patients with tender right sides, possibly with a segmentally filling appendix or one which retained the barium for many days. I observed them before and after operation. Many of them were apparently entirely cured by the operation but all too often the symptoms recurred after a period from three months to a year and the patient would again seek information why the abdominal symptoms persisted.

The old story—adhesions—is all too familiar. I would not attempt to estimate the number of people sent to our laboratory in an effort to locate adhesions in the caecal region as a cause of the renewed symptoms. The same is true of the gall bladder. Think of the number of individuals operated upon on meager histories and physical findings, indirect or indefinite x-ray findings. The gall bladder is found at operation apparently normal ,no adhesions, no thickening of the wall, no shrinking or distortion. The operative sheet usually states that the bile is very dark in color, very thick, quite tarry, or what not. The patient recovers from the operation, thanks to the good operative technique, and very often cured of his dyspepsia.

Here again, all too frequently, he returns after a varying period with a recurrence of his symptoms. He is again sent to the roentgenologist for a demonstration of upper abdominal adhesions. The next chapter may be an operation for the removal of the gall bladder and again the patient has a period of remission from symptoms, only to return later on. The next chapter may be one in which the genito-urinary specialist is consulted and a loose kidney or a kinked or strictured ureter diagnosed as the cause of the symptoms.

He may now try one of the cults in his frantic efforts to relieve his dyspepsia. He may become resignd to his fate and remain a chronic dyspeptic to the end of his days.

This picture is not overdrawn, I have seen it many, many times. I have in my records a large number of such histories, some even more prolonged and complex.

What is the answer to the question then? Why do some patients remain well after these operations and some have recurrences? The answer, in my opinion, in the vast majority of instances, is that the enforced physiological rest and dietary restrictions incidental to the operation cures or temporarily relieves the chronic colitis which is so often the cause of the symptoms in the first place.

I am led to this belief because I have successfully treated many such patients

without operation. I have also treated an approximately equal number with recurrent dyspepsia after operation with the same good results. After observing the almost universal relief from symptoms when the patients are given treatment directed to the colon, I am today more firm in my belief than ever before. Physiological rest of the colon carried out for a much longer period than is usually enforced after operation seems to me to be the key to the successful treatment of the vast majority of the chronic dyspeptics.

I have observed that the surgeon who kept his patients in bed a long time after operation and restricted the diet for a number of weeks or months, has a much smaller percentage of failures than the spectacular surgeon who is proud of his record of never keeping a patient in bed more than a week or ten days. The latter type of surgeon often gives his patients no advice as to his diet, in fact is quite happy to simply say, "Get up, go about your business, eat anything you want. I have removed the cause of your dyspepsia by taking out your appendix." And paradoxically as it may seem he very often gets away with it, the patient is relieved of his symptoms and swears about the miracles of modern surgery.

All too often, however, the symptoms recur. The patient suffered from too severe a grade of colitis and soon lapses into the same old dyspeptic syndrome again.

Let us now consider this disease from the standpoint of etiology, symptomatology and treatment. In this discussion I wish to exclude chronic amebic or bacillary dysentery, ulcerative colitis, tuberculosis, syphilitic stricture of the rectum, chronic obstruction of the bowel, tumors of the colon, diverticulosis of the sigmoid and the various parasitic diseases. It is assumed that the history and examination will exclude them.

I would like to say at the outset that the condition is not confined to the visceroptotic individual. I do not believe that the position of the abdominal viscera has much to do with it. I grant that these people do suffer more acutely and complain more bitterly when they are affected with the disease but that I think is due to their disposition to complain more about everything. It is common knowledge that a visceroptotic may be put to bed and fattened up and be relieved of all symptoms but another x-ray examination will show the viscera in exactly the same place as before the treatment. Furthermore roentgenologists know that the worst case of visceroptosis, with the stomach in the pelvis and the transverse colon making a "U" shaped dip to the floor of the pelvis, the sigmoid very redundant, may have absolutely no constipation, no retention beyond the normal limits and no dyspepsia. I find fully as many fat people suffering from colitis as I do the thin visceroptotic ones.

The story of the complaint is nearly always the same, varying only with the temperament and story telling ability of the patient. First there is a long period of constipation. This may date back as far as the patient can remember or may date from some bed confining sickness at which time the physic habit was formed.

I have made a habit of inquiring who was responsible for the development of the physic habit and all too often the patient states that it was begun on the advice of some physician. It has always been a remarkable thing to me that so many physicians prescribe physics to move the bowels, yet I am convinced that it still is a very common practice.

I think it is a very pernicious thing to prescribe physics of any sort to any patient unless it is justifiable occasionally in an acute infectious or toxic disease where there is actual need of removing poisonous material from the gastro-intestinal tract. Even then I feel sure it is greatly overdone, the patient instead of deriving benefit from it is further weakened and recovers more slowly than otherwise would have been the case. Physics may also become necessary in cardio-vascular or cardio-renal disease or for the individual who is too old or feeble to adopt proper habits of hygiene.

The constipation remains a simple constipation for many years. By that I mean there is very little local or remote symptomatology other than the fact the bowels will not move unless some physic is taken and the patient is subject to what he calls bilious attacks. After a time the particular physic becomes ineffective and a different kind is used. The patient usually goes through the entire list of physics, laxatives, regulators, tonic laxatives, aperient waters, mineral waters, mineral oils, etc. It makes no difference what the drug is or by what fancy name the pharmaceutical house gives it, the effect is always the same, the patient is never cured and nearly always becomes worse.

All such drugging is contrary to physiological laws and there comes a time when the delicate autonomic nervous system rebels. When this time arrives, the case is

no longer one of simple constipation but becomes something additional. The patient then begins to have chronic dyspepsia, begins to notice that certain foods do not digest well, that after eating them there is an uneasiness or actual distress in the abdomen. This is the beginning of the abnormal irritability of the bowel. There may be no actual macroscopic imflammatory change in the mucous membrane of the bowel but the delicate plexuses of Aurbach and Meissner located in the walls of the intestines and particularly those in the colon begin to show the strain of the long continued insult.

The normal mass peristaltic action of the large bowel begins to change to a segmental affair and one can see spasms at various points, particularly in the descending colon and sigmoid. The stools become small in caliber and are often passed in small balls like sheep dung. Later there may be alternate periods of constipation and diarrhoea. During the diarhoeic stage the stools are often putty like and are flat or ribbon shaped. The patient says he feels good only when he is constipated. Frequent attacks of fermentation occur accompanied by the passing of copious light yellow stools which float on the water in the toilet and have a very vile penetrating odor.

The principal subjective symptom is bloating after meals. Whenever a patient states that the abdomen feels unusually tight or distended shortly after meals it may be taken almost without exception that there is something wrong with the colon.

The two divisions of the para-sympathetic nervous system which control the motor activities of the gastro-intestinal tract are connected together in the higher brain centers and the only kind of stimuli which they can receive and transmit are those similar in nature to their normal function. Every sensory nerve is specific according to the nature of its end organ. The optic nerve because of the nature of its end organ is sensitive to light, and carries only sensation of sight. The auditory nerve because of the nature of its end organ carries only sensation to sound. If the optic nerve is stimulated by a blow on the eye the result is not pain but subjective sight sensation.

The sensory nerves of the gut carry sensations of pressure or stretching because their function is to contract the musculature of the bowel which they do under normal circumstances without pain or discomfort.

When the stimulus is abnormally severe the patient may suffer pain. If the stimulus is only moderately increased there may be only a feeling of bloating or distension. A measurement of the abdomen at these times may show no actual bloating, the colon is simply contracted down to a smaller calibre upon a normal amount of gas giving the patient a sensation of actual distention.

Whenever food is taken into the stomach it gives rise to the so-called gastro-colic reflex. In the normal individual this causes a desire to move the bowels usually directly after breakfast as this is the time the stomach has been emptied the longest. There is no pain, no bloating, only a desire for an evacuation. After the evacuation is completed the individual feels comfortable; feels that the bowel is empty; there is a sense of well being; a sense of stimulation, which was not present before.

This is not the case when the colon is irritated. The feeling of distension appears shortly after meals and is not confined to the morning meal, but may appear after any or all of the meals. Even a drink of water may bring it on. There is no particular desire at first to move the bowels and the bloating continues until after there is actual pain. The pain is colicky in nature and finally culminates in a large copious movement beginning as a hard mass continuing as a putty like flat stool and terminating as a watery one. The longer the action continues the worse the cramps become until at the end of the act the individual is in a cold sweat, feels weak and exhausted and may be forced to lie down for a time before the pain subsides.

This is the picture of severe chronic colitis. Of course one seldom sees such a neglected case. The majority complain simply of distension or uncomfortable fullness. They feel better after the clothing is lossened around the waist. This feeling may pass off after a time with the passage of some gas without a movement of the bowels.

My surgical friends will immediately say "Why this is exactly what we rely upon for a diagnosis of chronic gall bladder disease, all these patients complain of bloating after meals. We ask about this particularly and seldom do we operate a patient in which this is not present."

This is unquestionably true, most gall bladder patients have this symptom but I do not think it is due to the gall bladder directly but to the accompanying colitis which is so often present in chronic gall bladder disease. I think that bloating shortly after meals is the one symptom

which can be relied upon to tell us that the colon is disturbed.

The fact that chronic gall bladder disease may be present in almost any patient, with or without abdominal symptoms, makes it necessary to rule it out in every case. Formerly it was necessary to rely almost entirely upon clinical reasoning to diagnose chronic gall bladder disease but thanks to the dye visualization test it is now possible to rule it out in most instances, not however with 100 per cent accuracy.

The pain in colitis may be general over the entire course of the colon or may be confined to a certain segment. A characteristic feature is that the pain is inclined to shift from one part to another, appearing first on one side then the other, again in the epigastrium.

It varies from a simple feeling of distension or fullness to the most acute agonizing colicy spasm. When it is confined to the epigastrium it can easily be confused with an acute gall bladder attack if the history and physical findings are not carefully considered.

The caecum is nearly always distended and tender. The descending colon and sigmoid can usually be palpated as a hard tender mass.

The constant tenderness over the caecum often leads to a diagnosis of appendicitis. It is a good working rule that a constant tenderness over the right side is not appendicitis. To my mind the proof of this is that it may be present before or after an appendectomy and disappears only after instituting treatment directed to the colon. There should be no difficulty in differentiating colitis from appendicitis if we grant that chronic appendicitis does not exist except as a rare complication of tuberculous colitis or some such chronic granulomatous infection. Appendicitis is an acute or subacute disease with the usual symptoms of appendicitis present.

The remarkable thing about recurrent appendicitis is that so often the patient is perfectly well between attacks, does not suffer from dyspepsia and does not have tenderness over the right iliac fossa unless some complication exists. An x-ray examination made during the intervals between attacks may disclose a fixed caecum or a fixed retro-caecal appendix which is not particularly tender.

Sufferers from chronic colitis will nearly always state that such foods as raw apples, boiled cabbage, coarse vegetables and raw fruits of various kinds aggravate the symptoms. They learn to avoid these things of their own accord. They state that if they eat only a little food at a time and that of a simple carbohydrate or proteid nature they are free from pain. They say they feel better when constipated but that they are afraid to remain constipated too long and resort to a large soap suds enema or a physic to empty the gut. Then they begin to have distress again and after a period of food denial become constipated and are forced to the dynamiting process again.

Often books on diets are purchased and the patient will read that regular bowel movements can be secured by the proper combination of fruits and vegetables. He is urged to eat a large number of coarse vegetables each day; drink plenty of water; take a lot of exercise in the open air; go to stool at regular times each day; in fact the usual routine for simple constipation. This only aggravates the symptoms. The time to apply such a regime is before complications arise. An anti-constipation diet should never be prescribed if the history or examination discloses any evidence of irritation of the bowels. Many of the failures in the treatment of chronic bowel complaints can be directly attributed to a disregard of this fundamental rule.

X-ray examination of the gastro-intestinal tract as a rule will not disclose any characteristic thing. Often the roentgenologist's clinical sense will tell him what the trouble is even though he sees nothing abnormal. Sometimes the descending colon will be very spastic, but this is not diagnostic. Spasticity is so often noted in individuals who have no abdominal distress and no constipation and must therefore be interpreted cautiously. As a matter of fact the roentgenologist will make his diagnosis on the absence of organic stomach or gall bladder disease and the presence of pain along the course of the colon.

The sigmoidoscopic examination is probably the most important single aid aside from the history. The typical case will present characteristic changes. I have arbitrarily classified the cases into three groups. 1st, those that show no changes in the mucosa of the lower segment of the bowel within the range of the thirty cm. tube. These cases may have changes higher up, possibly in the caecum. I have had no opportunity to check up on this and know of no literature on the question. The lower segment, however, is free from macroscopic mucous membrane changes. There nearly always is a marked spasm of the gut at the

recto sigmoid juncture and considerable pain with the passage of the tube through this area. The recto-sigmoid juncture seems to act as a sphincter and the sigmoid will contain isolated, small hard fecal masses. This type of case seems to me to be a simple increased irritability of the colon with resulting spasticity.

The second class shows the same condition at the recto-sigmoid juncture plus a slight change in the mucous membrane of the lower bowel. There is a definite congestion of the blood vessels, a slight reddening of the mucosa and that is all.

The third class, and the one so commonly found presents a very characteristic picture. The mucous membrane is fiery red, definitely granular and shows a tendency toward redundant folds. The bowel does not baloon out ahead of the sigmoidoscope, the patient complains of pain the very minute the instrument is inserted through the anal sphincter and the examination can rarely be carried higher than the recto-sigmoid juncture on account of the pain. The wall of the gut may be covered with small sticky fecal masses. The mucous membrane bleeds at the slightest touch but there are no erosions or ulcerations present. The anal canal may contain shallow fissures and small hemorrhoids and after the examination the patient feels that he has been through a real ordeal.

I have spoken a number of times about physiological rest of the colon as the particular therapeutic agent necessary to accomplish a cure. Just what is meant by that? If one has a sprained or bruised elbow the arm is put in a sling and after a few hours the injured nerve endings quiet down and the pain subsides. This principle applies to inflammation anywhere—rest and the inflammation subsides. Exactly the same principle is applied in treating colitis.

In acute colitis we limit the food intake to almost nothing, give opiates and bismuth, and the patient feels better very shortly. In the treatment of a chronic disease the same principle is utilized but not to such an extreme as in the acute disease. In a disease which may require months to cure one cannot limit the food intake below the maintenance level. Neither is it justifiable to give opiates over a long period of time. The treatment then must be such as will fulfill the requirements of a maintenance diet and still obtain the maximum amount of physiological rest for the colon.

An inquiry into the nature of foodstuffs discloses the fact that certain foods have a tendency to move the bowels, either by the bulk of their residue or by the amount of irritation and fermentation they produce. If such a diet can be prescribed that will fulfill the patient's food value needs and at the same time not produce fermentation, irritation or undue residue, the desired results are obtained.

Acting upon this line of reasoning we immediately single out the vegetables as the principal bulk producing food and also as one of the principal fermentation offenders. Fruits come under the ban on account of the fermentation they produce. Such irritating foods as pickles, olives, condiments, jams, jellies, candies, soda fountain drinks, pancakes, hot breads, pork, fried foods, very hot or cold foods, cider, buttermilk, strong coffee or tea, alcoholic liquors, rich pastries all come under the ban.

This leaves then a large list of easily digestible foods, with practically no residue and only a mild tendency toward fermentation or irritation. The list contains—

Soups—creamed, strained vegetable or bouillon.

Cereals—All cereals except bran foods. All should be well cooked.

Bread—Stale bread, toast, zwiebach and crackers.

Meats—All meats and poultry except pork (crisp bacon is allowed in moderation.)

Dairy products—Milk, cream, butter and simple cheese.

Eggs—Any way except fried or deviled.

Miscellaneous—Spagetti, macaroni, (no tomato sauce), rice, tapioca, noodles.

Desserts—Jello, custard, cornstarch, ice cream, ices, bread pudding, very simple cake.

Fruits—None except a little orange juice night and morning.

Vegetables—None.

This non-residue, non irritating diet will fulfill the proper food requirements, in fact, it is highly nutritious and patients often get fat on it. At the same time it is practically all absorbed before it reaches the colon and threfore the colon has very little to do and practically nothing to irritate it. . Such a diet is constipating and it is nearly always necessary to give a two or three ounce cotton seed oil enema at night which is retained until morning. This will insure a bowel movement the next morning if there is anything in the lower bowel to be removed.

It is sometimes necessary, if the bowels are very loose, to give a powder consisting of equal parts of calcium carbonate, cal-

cium phosphate and bismuth subnitrate in teaspoonful doses three or four times a day. Many times small doses of opium and belladonna are helpful at the start, the balladonna may well be continued for several weeks. It is often necessary to put the patient to bed and apply heat to the abdomen several hours a day, preferably with the electric pad. The weight of a hot water bottle is often very disagreeable to the patient.

Local treatment applied to the mucous membrane of the lower segment of the bowel is a very great aid and sometimes apparently essential. A very helpfull remedy is to insufflate through the sigmoidoscope daily a powder composed of equal parts of calomel and bismuth subnitrate. This is plastered all over the wall of the gut and it is surprising how quickly the inflammation subsides following its daily use.

Under this plan of treatment the patient will show improvement at once and the improvement will continue steadily from week to week. A mild case may need the strict diet only a few weeks to effect a complete cure. This to my mind accounts for the cures when a normal appendix is removed under the assumption that it is the cause of the trouble and also accounts for the failures if the inflammation is too severe.

A severe case may require several months before the dietary rstrictions can be removed. The criterion for increase in diet is a complete relief from pain and bloating or irritation of the bowel as determined by frequent sigmoidoscopic examinations.

When increases are made in the diet, one should begin with the simple vegetables such as spinach, string beans, mashed or baked potatoes, hearts of celery, peas etc. Stewed fruits such as apples, prunes, peaches, apricots, etc, may be added cautiously as the patient's tolerance permits.

At times it may be necessary to continue the restrictions on raw fruits and coarse vegetables for one or two years and the patient can usually be promised that in time a normal diet can be resumed, subject to modification if there is the least return of symptoms. The average patient who has suffered from dyspepsia for years will gladly submit to a reasonable restriction in diet after he has been shown that such restriction keep him comfortable.

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In the last number of the Illinois Medical Journal the Editor severly criticizes Dr. Arthur Dean Bevan for a statement he is reported to have made at the annual conference of the council on medical education and hospitals of the A. M. A. Newspapers quoted Dr. Bevan as saying:—"More than ninety-nine prescriptions out of a hundred written for a pint of whiskey are bootlegging prescriptions and are a disgrace to the great medical profession."

Men who have achieved greatness in the medical profession are too frequently regarded as infallible critics of medical thought and medical practices, by the profession as well as the laity. It is to be expected that they should overestimate the importance of their opinions and present such opinions as facts. The profession in Kansas cannot be included under Dr. Bevan's arraignment of the profession as bootleggers. That is, at least, one advantage in the bone dry law.

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Silver Compounds

Silver nitrate first demonstrated the bactericidal property of silver. This was, obviously, an invitation to the chemist to devise a silver compound that could be used freely in solution, as silver nitrate could not. Especially desired was a silver salt that would kill the gonococcus without irritating the urethra, for it was soon learned that silver was especially efficacious as a gonococcide. The majority of silver compounds offered from time to time one of two objections is made by the patient: first, they hurt; second, they left dark stains on the linen.

Now comes a comparatively new silver iodide preparation—one that actually protects the silver and the iodine from the action of light, and yet leaves its activity as a germicide apparently unimpaired. Neo-Silvol, as this product is called, is said to be 20 times as active as pure carbolic acid (in other words, to have a phenol coefficient of 20) in contact with the gonococcus, and at the same time to be notably bland in its effect upon the inflamed tissues and free from the dark-staining tendency that characterize other silver preparations.

Further particulars are offered to the readers of the advertisement on Neo-Silvol which appears on another page of this issue.

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A copy of the "Preliminary Report of the Commission on Medical Education" will be sent to any one interested in the general qustions of medical education and practice. Address: Commission on Medical Education, 215 Whitney Avenue, New Haven, Connecticut.

THE JOURNAL
of The
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THE MEDICAL SCHOOL

The recent legislature appropriated $300,000 for new buildings for the Medical School, marking an epoch in the development of this institution. This will provide for a nurses' home accommodating eighty nurses and for a model ward unit with a capacity of 120 beds. This appropriation is of more significance than the mere addition of two badly needed buildings. It means that the state has endorsed the present plant and has put itself on record as favoring the development of a modern medical school for Kansas. It shows that the state is alive to the importance of a properly supported medical school in safeguarding the health of its citizens. It indicates a more consistent adequate support of this institution in the future.

Another act of the same legislature promises a most important part in the future development of the Medical School. This was the provision which separated the budget of the plant in Kansas City from Lawrence without separating the two divisions, thereby insuring University control and supervision. This should make it easier for both the University and the Medical School to receive more adequate support from the legislature for both buildings and maintenance.

The Medical School owes much of these recent developmnts to the wholehearted support which it received from the medical profession of the state. It is only natural that it should, in its development, justify the faith and support that the physicians of the state have given it. This can best be accomplished by consulting the medical profession in developing its plans for the future, by explaining its policies, by inviting criticisms from over the state, and by welcoming suggestions on means of enhancing its service to the medical profession of the state by postgraduate instruction, diagnostic service, and so on.

The school is anxious to cooperate with the medical profession of the state especially with some perplexing and intimate problems that face it. Inasmuch as it is supported by all of the people of the state there are demands made upon it by laymen that cannot be ignored. These often involve the general practitioner's field and for this reason the cooperation of the profession at large is essential. This is illustrated in the increasing recognition of the right of the average wage earner to the best the medical profession has to offer at a cost that he is able to bear. The demand of society that each crippled child be given whatever medical science has to offer regardless of its financial backing has lead many well meaning but often misguided individuals to take forcible measures even against the advice of the doctors. This is often associated with a general apathy on the part of the medical profession so that ill-advised health legislation may be forced upon the physician. In some states there seems to be an increasing tendency for health measures to be under the supervision of lay individuals and entirely independent of the medical profession. This is a dangerous tendency and one that the School desires to counteract. It would encourage only such general health measures as are under direct supervision of the medical profession and its policy in reference to the

care of crippled children and in othr measures is shaped with this end in view of giving this child the proper attention it requires, but to do it through the local profession and if possible aid the local doctors to carry this out themselves.

The Medical School has established clinics for crippled children in different sections of the state in order to give these children a service that heretofore they have not been able to get. These clinics are developed only on request of the local medical society to the School that this be done. All local arrangements for securing the clinical material are under the control of the local physicians. Only those patients who cannot afford to secure a physician to care for them and those who are sent or brought in person by their family physician may attend these clinics. The Medical School sends a member of its Orthopedic Department to these clinics once a month to take care of those whom the local medical group have assembled together. These clinics are being held in Hutchinson, Pittsburg, Dodge City and a new one is soon to be established at Ellsworth or Hays. They have been carried on a little over a year and over two hundred crippled children have received attention who would otherwise have been neglected. This is the answer of the Medical School and the medical profession to the growing demand of the people of the state that these unfortunate children be given every opportunity to overcome their physical handicaps. It has not only helped these children but has done so through the local physicians with the assistance of lay organizations.

The policy of the Medical School to cooperate with the profession of the state makes it unwilling to enter into competition with any practitioner. This is especially true of the specialties, such as the x-ray. Owing to the fact that the School is supported by the state it is often faced with delicate situations, for example, that of giving a service that it can give and a citizen of Kansas badly needs, but one which belongs to a doctor in the town where the patient lives. Such a patient is advised to return to his doctor but he often insists that he does not want him any more and, inasmuch as he pays taxes, that the School must take care of him. This is done but he is charged the usual professional fee unless his doctor signs a statement that he is unable to do so. Patients often deny that they have a doctor and the School is at times imposed upon by them. An effort is made to prevent this if possible. It is for this reason that patients who are sent in for special examination and treatment without a written statement from their doctor frequently are put at some inconvenience until they can be consulted by the Superintendent of the Hospital to see if they are legitimate cases for this service. Prompt and adequate service is insured if the doctor sending the patient communicates with the hospital beforehand, or sends a written statement with the patient especially noting what is wanted and whether the patient can pay the usual fees. Even then delays occur because the Hospital is usually crowded with patients.

There is an active free dispensary in the School. Many patients from over the state who can afford to see a private physician come to this clinic. An effort is made to discourage them but as a state institution this is very difficult to enforce. A man states he has no money and it is too expensive for the School to investigate his case; hence he is taken at his word and treated accordingly.

There is some misconception among physicians regarding the Hospital. Many seem to have the impression that because it is a state institution that it has many free beds and is supported by the state. There are no free beds. The Hospital must secure its support from the fees that it collects from patients. Most of the beds are ward beds for which $15.00 is charged per week. There are many free patients but these patients are county patients whose hospital bills are paid by the counties in which they reside. Moreover, the Hospital beds are almost always filled so that no patient should be

sent in without a reservation being made in advance.

There is another field where the School is planning to be of assistance to the members of the profession in the state. The progress of modern medicine is becoming so rapid that it is impossible for the average busy practitioner to sift out the important advances and make use of them without taking repeated postgraduate courses every few years. For the average man this is impracticable not only because of the expense but also because of the interruption of his practice that it entails. The School is planning on bringing the postgraduate instruction to his door following the plan that first became popular in North Carolina and has recently proved successful in Oklahoma. The School is at present seeking advice from the profession to see how many are interested in such a course. If a sufficient number manifest a desire for it, it will probably be offered in the next year.

The policy of the School is to be of service to both the state and the medical profession. It emphasizes the spirit of service to each of its graduates and is ready to assist them in rendering this service after they enter practice. The same assistance will be gladly given to any reputable practitioner of the state. Any suggestions or criticisms regarding the work of the Medical School are always welcome for from them its service is often made more efficient.

H. R. WAHL, M.D.

MEDICAL SCHOOL NOTES

Dr. Willis C. Campbell, Professor of Orthopedic Surgery, University of Tennessee College of Medicine, talked to the Senior Class, Tuesday, March 29.

Dr. Sam H. Snider addressed the students of the Kansas City Teachers' College, on the subject "Tuberculosis," Monday, March 28.

Dr. Richard L. Sutton was the guest of the President and Faculty of the University of Missouri. He gave a natural science lecture in Jesse Hall, in the evening, and while at Columbia also lectured at Christian College and Stephens College.

Dr. C. C. Dennie talked over the Star Radio on the subject, "The Evolution of Electricity in the Treatment of Disease," March 30.

Dr. Richard L. Sutton was the guest of the Saline County Medical Society at a dinner and meeting on March 16th. In the evening he gave a public lecture at the Marshall High School.

Dr. Pearl Matthai, of the class of 1926, has accepted an internship at the Los Angeles Childrens' Hospital, Los Angeles, California.

Dr. E. T. Gibson addressed the Girls' Health Club at the Paseo High School, April 7. Dr. Gibson's subject was "Mental Hygiene in the New Public Health."

At the meeting of the American College of Surgeons, March 28, 29, the following members of the Faculty took part on the program: Dr. C. B. Francisco, Dr. John G. Hayden, Dr. Thomas G. Orr, Dr. J. L. Meyers, Dr. Sam E. Roberts, Dr. L. F. Barney, Dr. N. F. Ockerblad, Dr. Frank R. Teachenor.

Dr. I. J. Worf spoke over the Star Radio on the subject, "Only A Cold," April 6.

At the recent election of the Academy of Medicine Dr. L. F. Barney was elected President, Dr. J. G. Hayden, Vice President, and Dr. F. C. Helwig, Secretary. Dr. P. T. Bohan was appointed a member of the Executive Committee.

Dr. Harry M. Gilkey of the Pediatrics Department has been granted a leave of absence to study in a childrens Hospital in Berlin.

DEATHS

D. R. Stoner, Ellis, Kansas, aged 47, died suddenly while assisting in removing furniture from a burning dwelling, April 17th, 1927. Dr. Stoner graduated from University Medical College, Kansas City in 1905. He has been for a number of terms councillor for the tenth district.

Henry M. Hall, Lincoln, Kansas, aged 91, died March 20th at Rochester, Minn. He graduated from the College of Physicians and Surgeons, Keokuk, Iowa, in 1858.

Zachariah Nason, Kansas City, Kansas,

aged 72, died February 20th, of retroperitoneal sarcoma. He graduated from the College of Physicians and Surgeons, Baltimore, in 1888; was formerly professor of obstetrics in the University of Kansas School of Medicine and was on the staff of Bethany Hospital. He was a member of the Society.

Dr. John Cornelius Entz, Salina, aged 54, did February 7, 1927, of pernicious anemia. He graduated from Eclectic Medical College, Cincinnati, Ohio, in 1896.

SOCIETIES

DECATUR-NORTON COUNTY SOCIETY

The Decatur-Norton County Medical Society met at the State Sanitorium for Tuberculosis on April 6, 1927 at 10:00 a.m. and the following program carried out. Two incipient cases of tuberculosis in children were presented to the society by the sanitorium staff, and discussed, and x-ray plates shown.

At noon, luncheon was served to the physicians and their wives.

At 2:00 p. m. the business meeting convened and the following program was given A case of mitral insufficiency in a young child, and a case of endocarditis in a woman 60 years of age were brought, by Dr. Ben Morris and Dr. I. L. Parker, respectively.

An excellent paper on malignant endocarditis was read by Dr. C. C. Fuller, which was discussed by Dr. Kenney, Dr. Boughnou and Dr. Parker.

Dr. H. P. Boughnou of Kansas City then read a splendid paper on coronary occlusion, which was well received by the society and was generally discussed by several of the members. He presented a different side of what was supposed to be a rather unusual condition; but from his description it occurs much more frequently than was formerly thought.

Upon motion by Dr. Lathrop, seconded by Dr. Piper, Dr. Boughnou was voted in as an honorary member, with all the privileges of the same.

A letter from Dr. Sterrett of Hutchinson was read, inviting all golf players to be at Hutchinson if possible on Monday, May 2nd, for a doctors' golf tournament. .

At the same time that the doctors were in session the ladies' auxiliary was organized. It is undrstood that the ladies will meet frequently at the same time the physicians do.

The doctors and wives were entertained at 6:30 dinner at the Kent hotel by the local physicians.

The following members and visitors were present: Dr. H. Boughnou, K. C., Dr. V.. C. Eddy, Dr. C. W. Cole, Dr. J. H. A. Peck, Dr. H. O. Hardesty, Dr. I. B. Parker, Dr. Benj. Morris, Dr. Demay, Dr. Walter Stevenson, Dr. G. A. Leslie, Dr. R. M. Tinney, Dr. C. S. Kenney, Dr. H. M. Hostetler, Dr. C. C. Fuller, Dr. E. L. Morgan, Dr. F. D. Kennedy, Dr. W. C. Lathrop, Dr. F. R. Funk, Dr. H. S. Bennie, Dr. F. E. Gaither, Dr. W. J. Lowis, Mr. F. E. Walton, Omaha, Dr. Lottie F. Law, Dr. D. H. Morgan, McCook, Dr. J. Jeurinck, Dr. M. J. Renner, Dr. F. H. Smith, Dr. W. F. Deal.

C. S. Kenney, M.D., Secretary.

MISSOURI-KANSAS NEUROPSYCHIATRIC SOCIETY

The Missouri-Kansas Neuropsychiatric Society met at State Hospital No. 2, St. Joseph, Missouri, April 13, 1927.

Dr. T. H. Romeiser, presented a case of late encephalitis epidemica, with a torsion spasm.

Dr. H. O. Daniel presented a case of parkinson syndrome, following encephalitis.

Dr. J. H. Parker presented a case of multiple sclerosis, with spastic paraplegia.

Major Edgar King, U. S. A. Disciplinary Barracks, Ft. Leavenworth, read a paper on "The Commoner Mental Defects and Disorders, and their Detection in the Selection of Recruits."

E. T. Gibson, Secretary.

SEVENTH DISTRICT SOCIETY

The Seventh District Medical Society enjoyed another of its meetings at Sterling on Wednesday afternoon and evening, April 13. On account of theatening weather the attendance was not large, but a profitable program was enjoyed by those fortunate enough to be present.

Dr. Alfred O'Donnell of Ellsworth gave a paper on "Some Observations on Diseases of the Biliary Tract."

Dr. F. L. Dennis of Dodge City, on "Mistaken Etiology and Treatment of Hay Fever.'

Dr. A. E. Bence of Wichita, on "Low Backache." Dr. Bence had car trouble and did not arrive till the dinner hour, but all were glad he was able to be with us, and his paper, as well as each one given, was thoroughly enjoyed and well discussed.

The Sterling fraternity entertained the society at dinner and the Music and Ex-

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The Diagnostic Department of Research Hospital was established in November 1924. Patients are received for diagnosis from reputable physicians. On completion of examinations, reports, which include the patient's history, physical examination, laboratory and x-ray reports, the findings of various specialists and the final diagnosis with recommendations for treatment, are sent to the patient's physician—in no instance will reports be given to patients. The fee includes all necessary tests and examinations. The following Departments are represented:—

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pression Departments of Sterling College furnished after-dinner entertainment with a Male Quartette and with a one-act play entitled, "The Dear Departed," which pleased all.

Officers elected for the coming year are: Dr. R. W. Springer, Kingman, President; Dr. M. C. Jenkins, Pratt, Vice President; Dr. H. R. Ross, Sterling, Sec.-Treasurer.

The fall meeting will be held at Larned in October.

H. R. Ross, Secretary.

WILSON COUNTY MEDICAL SOCIETY

The Wilson County Medical Society met at Neodesha Friday evening, April 22d. Dr. Earle G. Brown, Secretary of the State Board of Health and President of the State Society was the guest of honor and speaker of the evening.

Dr. Brown spoke for an hour on the organization of the State Board of Health and birth and death reports and other things. The talk was very interesting and threw light on the functioning of the State Board.

Dr. C. H. Kinnaman, State epidemiologist, talked on diphtheria toxin-antitoxin and one could see he is an enthusiast on the subject.

The discussion was general; Dr. Brown remarking that we should be careful and de-sensitize when in doubt.

The President, Dr. W. H. Young, then called on Dr. C. O. Shepard of Independence and Dr. Ross of Sterling, guests, who responded with short interesting talks.

This society was much pleased with the speakers and want to thank them for an enjoyable and profitable evening.

Adjourned to meet in May.

E. C. Duncan, Secretary.

SUMMER CLINICS, CHICAGO MEDICAL SOCIETY, 1927

Announcments and schedules will soon be ready for the 1927 Summer Clinics of the Chicago Medical Society, supported by many of the largest hospitals in the city, among them being the Post Graduate Hospital, Chicago Memorial Hospital, University of Illinois College of Medicine, Cook County Hospital, Michael Reese Hospital, Mercy Hospital, Presbyterian Hospital, Jackson Park Hospital, St. Luke's Hospital, Ravenswood Hospital, Mount Sinai Hospital, Francis Willard Hospital, West Suburban Hospital, Evangelical Hospital, North Chicago Hospital, Chicago Lying-in Hospital, St. Joseph Hospital, Alexian Brothers Hospital, Laboratory of Surgical Technique, Washington Park Hospital, Jackson Park Hospital, Chicago Municipal Tuberculosis Sanitarium, John B. Murphy Hospital. Several of our large laboratories have also agreed to co-operate with us in this great work.

In 1926 we limited registrations to physicians living in Illinois, but our increased facilities make it possible to accomodate many more than last year. Registrations therefore will be open to physicians from other states and to as many as may be accomodated, in the order of their registrations. Registration fee will be $10 for each two weeks course, payable at time of registration, and a physician may register for only one course of two weeks.

Admission will be by card only, issued by the Chicago Medical Society and no registration card will be issued until registration fee is paid.

The first two weeks course will begin on Monday, June 13th, 1927, at 9 a. m., ending Friday, June 24th.

The second two weeks course will begin on Monday, June 27th at 9 a. m., ending Friday, July 8th..

This is an excellent opportunity for the medical men of the country to obtain real post graduate work in some of the best hospitals in the world, and from some of the best clinicians found anywhere.

Schedules will be sent to the 10,000 physicians in Illinois, and announcements will be sent to the American Medical Association, and the several state medical journals.

Dr. Lawrence Nelson '19, visited in Kansas City, Kansas a few weeks ago.

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WANTED—We have several well-trained practical laboratory technicians graduating from our school of public health May 15; physicians, hospitals and health departments desiring such service can secure it by writing immediately; first come, first served. Address, Dr. L. H. South, State Bacteriologist, 532 W. Main St., Louisville, Ky.

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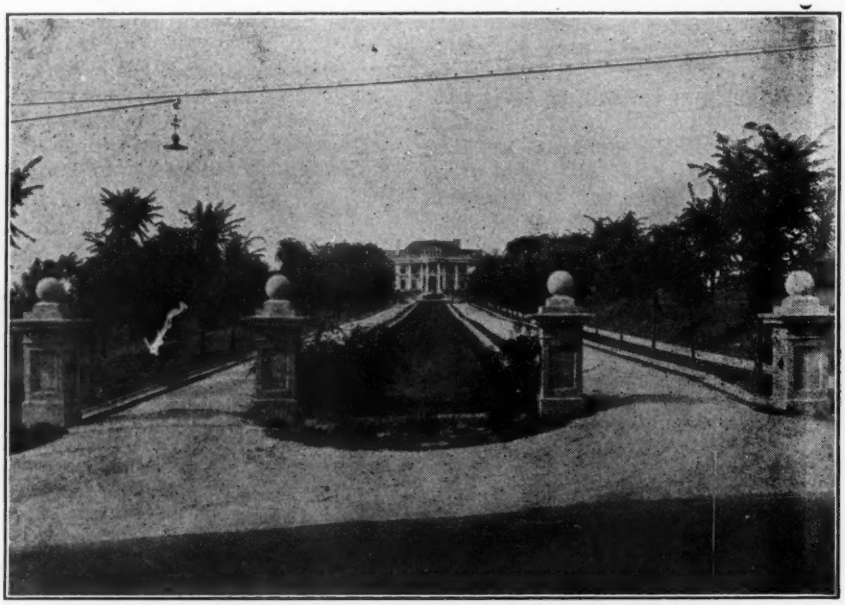

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(City and State)

graduated in the year 1_____and received the degree of_____

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(Name of Medical College)

_____located at _____

from which I graduated in the year 1_____

4. My state certificate was issued_____
(Name of state and date of license under which you are practicing)

5. I have practiced at my present location_____years; and at the following places for theyears named _____
(Name each location and give dates)

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_____

6. I hold the following positions:_____
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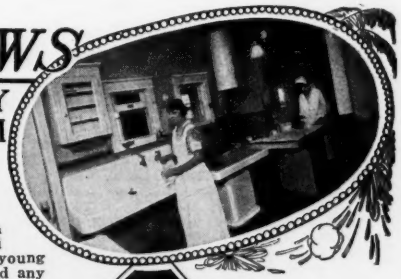

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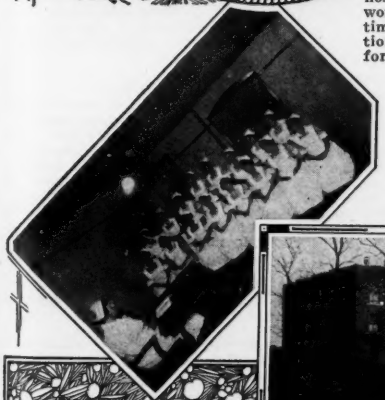

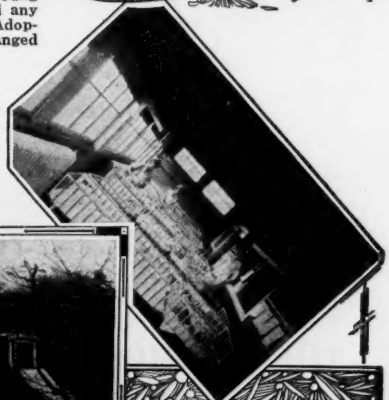

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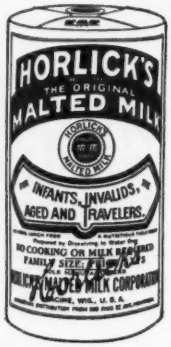

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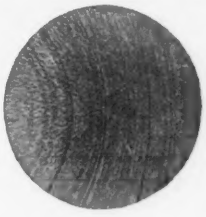

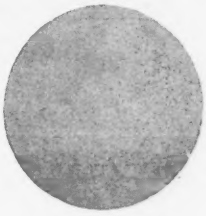

Left: Scarred surface of an ordinary lens.

Right: Tillyer lens surface.

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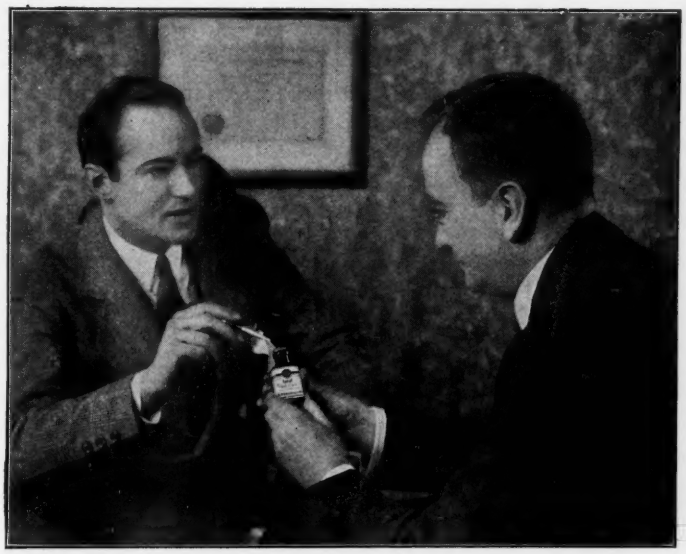

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KANSAS MEDICAL SOCIETY

Chartered by the Territorial Legislature of Kansas, February 19, 1859

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Members of Component County Societies are members of the Kansas Medical Society. Physicians residing in counties where no County Society exists may join the society of an adjoining county. Physicians residing where no County Society exists, who are members of a district or other independent society approved by the Council, may be admitted to membership.

ANNUAL DUES $5.00, due on or before February 1st of each year.

Dues should be paid to the Secretary of the Component County Society, or, if not a member of a County Society, to the Secretary of the Kansas Medical Society.

OFFICERS FOR 1926

COUNTY	PRESIDENT	SECRETARY	MEETINGS HELD
Allen		P. S. Mitchell, Iola	
Anderson	A. J. Turner, Garnett	A. J. Turner, Garnett	2nd Wednesday
Atchison	W. F. Smith, Atchison	S. W. Connor, Atchison	1st Wed. ex. July and August
Barton	H. W. Jury, Claflin	M. Morrow, Great Bend	1st Tuesday, Jan., Apr., June, Oct.
Bourbon	C. L. Mosley, Fort Scott	W. S. Gooch, Fort Scott	2nd Monday
Brown	W. G. Emery, Hiawatha	R. T. Nichols, Hiawatha	2nd Friday
Butler	J. C. Bunten, Augusta	L. L. Williams, El Dorado	2nd Friday
Central Kansas	J. B. Carter, Wilson	H. S. O'Donnell, Ellsworth	Dec., March, June, Sept.
Cherokee	R. C. Lowdermilk, Galena	W. H. Iliff, Baxter Springs	2nd Monday
Clay	X. Olsen, Clay Center	G. B. McIlvain, Clay Center	2nd Wednesday
Cloud	Andrew Struble, Glasco	R. E. Weaver, Concordia	Last Thursday
Coffey	H. T. Salisbury, Burlington	A. B. McConnell, Burlington	
Cowley	H. H. Jones, Winfield	J. R. Wentworth, Ark. City	1st Tues. ex. July, Aug., Sept.
Crawford	J. G. Conley, Pittsburg	Oscar Sharp, Pittsburg	3rd Thursday
Decatur-Norton	J. A. H Peck, St. Francis	C. S. Kenney, Norton	Called
Dickinson	E. J. Reichley, Herington	L. A. O'Donnell, Chapman	
Doniphan		W. M. Boone, Highland	1st Tues. Jan., Apr., July, Oct.
Douglas	F. R. Keith	J. B. Henry, Lawrence	1st Thursday
Elk	R. C. Harner, Howard	F. L. Depew, Howard	Called
Finney	R. M. Troup, Garden City	O. W. Miner, Garden City	
Ford	C. E. Bandy, Bucklin	W. F. Pine, Dodge City	Last Wednesday
Franklin	Geo. W. Davis, Ottawa	J. R. Scott, Ottawa	
Harper		M. B. Flowers, Harper	3rd Wed., Mar., June, Sept., Dec.
Harvey	R. C. Porter, Newton	H. M. Glover, Newton	1st Monday
Jackson	M. S. McGrew, Holton	C. A. Wyatt, Holton	1st Wed., Jan., Apr., July., Oct.
Jewell	J. E. Hawley, Burr Oak	C. W. Inge, Formoso	
Johnson	C. H. Lester, Olathe	D. E. Bronson, Olathe	
Kingman	R. W. Springer, Kingman	H. E. Haskins, Kingman	2nd Thursday ex. summer months
Labette	O. H. Ball, Dennis	R. W. Wrie, Parsons	4th Wednesday
Leavenworth	S. L. Axford, Lansing	J. L. Everhardy, Leavenw'th	1st Monday
Lincoln		M. Newlon, Lincoln	2nd Thursday
Linn	J. R. Shumway, Pleasanton	H. L. Clarke, LaCygne	2nd and 4th Fridays
Lyon	M. T. Capps, Emporia	J. A. Woodmansee, Emporia	1st Tuesday
Marion		E. H. Johnson, Peabody	2nd Wednesday
Marshall	J. L. Hausman, Marysville	J. W. Randell, Marysville	Last Thurs., July, Oct., Jan., Apr.
Meade-Seward	F. W. Huddleston, Liberal	B. H. Day, Liberal	
Miami	P. T. Gatley, Louisburg	J. W. Kelley, Louisburg	
Mitchell		E. E. Brewer, Beloit	
Montgomery	E. C. Wickersham, Ind	J. A. Pinkston, Independence	2nd Friday
McPherson		G. R. Dean, McPherson	
Nemaha	D. H. Fitzgerald, Kelly	S. Murdock, Jr., Sabetha	Last Thursday every other month
Neosho	G. Ashley, Chanute	J. N. Sherman, Chanute	Second Monday
Osborne	J. E. Hensnall, Osborne	S. J. Schwaup, Osborne	
Ottawa	J. D. Vermillion, Tescott	J. F. Brewer, Minn	
Pawnee		A. E. Reed, Larned	
Pratt		E. H. Ireland, Coats	2nd Tuesday
Reno	Irl E. Hempsted, Hutchinson	C. A. Boyd, Hutchinson	1st Monday
Republic	L. O. Nordstrom, Belleville	H. D. Thomas, Belleville	4th Friday
Rice	H. R. Ross, Sterling	C. E. Fisher, Lyons	2nd Thursday in November
Riley	A. H. Bressler, Manhattan	J. T. Mathews, Manhattan	Last Thursday
Rush-Ness	L. A. Latimer, Alexander	E. N. Sulis. McCracken	2nd Monday
Saline	C. M. Fitzpatrick, Salina	E. G. Ganoung, Salina	
Sedgwick	H. F. Hyndman, Wichita	W. J. Ellerts, Wichita	2nd Thursday
Shawnee	J. L. Lattimore, Topeka	E. G. Brown, Topeka	1st and 3rd Tuesday
Smith	O. C. Reed, Kensington	H. Haecle, Athol	1st Monday
Stafford	T. W. Scott, Stafford	J. T. Scott, St. John	Called
Sumner	A. R. Burgess, Wichita	W. H. Neel, Wellington	2nd Wednesday
Washington		W. M. Earnest, Washington	Last Thursday every quarter
Wilson	J. L. Moorhead, Neodesha	E. C. Duncan, Fredonia	2nd Monday.
Woodson		M. S. Reynolds, Yates Center	
Wyandotte	T. L. Richmond, Kansas City	H. W. King, Kansas City	Every 2nd Tues. ex. summer month